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OF THE

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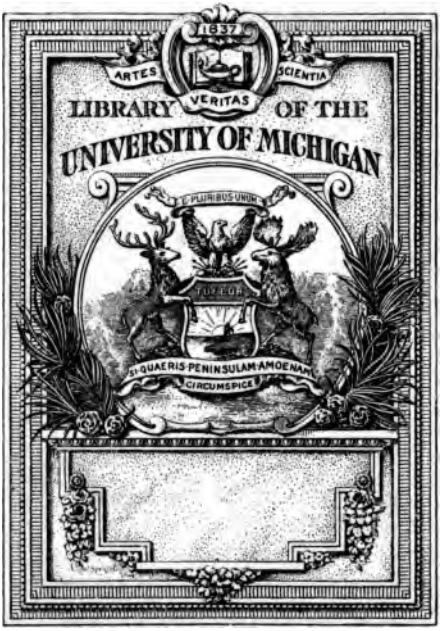
1905



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TRANSACTIONS

OF THE

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OF THE

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1905

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[Reprinted from THE MEDICAL NEWS, July 22, 1905.]

THE MEN WE LOST.[1]

BY S. J. MELTZER, M.D.,
OF NEW YORK.

THE remarks, which custom requires of me to make at this occasion, will be confined, as at last year's meeting, to matters pertaining to the affairs of the Association. Last year your president advocated a widening of the scope of the Association, and recommended the appointment of a committee which should formulate appropriate changes in the constitution. The recommendation being approved by a vote of the members, the president named seven members to serve on that committee which was termed Committee on Reorganization. After due deliberation and consultation with the members of the council, the committee recommends changes in the constitution which in the first place would enable the association to include in its membership scientific investigators as well as surgeons who do special work in gastro-enterology; and in the second place would make an irreproachable moral standing an indispensable requisite for acquiring and retaining membership in this Association. The council recommends the adoption of these changes. The report of the committee will be read to you at the executive meeting. According to the constitution, these changes could not be voted upon until next year. However, a part of the desired aims could be already realized the coming year if the president should be empowered to continue the present Committee on Reorganization for the purpose of studying the men who appear to be very desirable as members of our Association and to invite them to join our ranks. The present occupant of the chair would be very gratified if he could thus contribute to the elevation of the scope and standard of this Association.

In the address of last year I was happy to be able to point out that we never met yet with the

1 President's Address, delivered at the opening of the eighth annual meeting of the American Gastro-Enterological Association, held at the Academy of Medicine, New York, April 24 and 25, 1905.

flag at half mast. We still have reason to be thankful to the fates for the integrity of the ranks of our active members. I have, however, to record with profound regret that in the course of the past year one of our foreign members, a master mind in our science, was taken from us. I refer to Franz Riegel, who, through some inexplicable error, has been classed only among the corresponding members of this Association. Furthermore, in looking over the list of our foreign members, which originally comprised seven celebrated names, I found that nearly one-half of them have already been taken from us by death. Besides Riegel, fate has taken from us Adolph Kussmaul and Samuel Fenwick. There is no record that at the meeting of 1903 due notice was taken of these bereavements which occurred in 1902. It was an oversight which it is not now too late to correct. While it is within the scope of my duties to speak to-day of the life of Franz Riegel, I am sure the Association will grant me the privilege to speak also of the lives and works of the other great men who honored us during their life by accepting the membership of our Association.

Adolf Kussmaul was the first of our losses. He died May 28, 1902, at the age of eighty, of heart failure, half an hour after he experienced some stenocardiac symptoms for the first time. He was born in Graben, near Carlsruhe, Germany, as the son of a country practitioner from whom he inherited the faculties of a naturalist and the love for medicine. After completing his medical studies in Heidelberg and taking some post-graduate studies in Prague and Vienna, he served for a short time as military surgeon in the Baden army and then settled in Kadern, a small town in the picturesque region of the Black Forest, where he carried on a successful practice for four years until he became incapacitated for many months by an attack of paraplegia of obscure origin. After recovering from this disease he decided to begin life anew and to prepare himself for an academic career. With his meager savings as a base he pursued the study of the scientific branches of medicine in Würzburg. In 1856 he was admitted to the University of

Heidelberg as Privat-Docent and in the following year he was appointed professor extraordinarius. In 1859 he was called to the regular chair of medicine in Erlangen; in 1864 he went to the University of Freiburg and in 1876 he was called to the University of Strassburg. In 1888 he retired from his academic positions and spent the remainder of his life in his beloved Heidelberg.

Kussmaul was undoubtedly one of the greatest clinicans, one of the most accomplished physicians of his age. He was a harmonious personality; in him the great faculties of the naturalist and the physician were coupled with kindness and simplicity of heart, and in him were united the common sense of the practical man with a fine poetical spirit flavored with a delicious humor. He was admired and loved. His scientific contributions are too numerous to be mentioned at length here. They extend into nearly every department of medicine. Many of his contributions exerted a deep influence upon the science and practice of medicine, and some became common property to such an extent that their originator is already forgotten. Some of his writings are real classics. I mention only the essay on the Psychology of the Newborn or the important essay on the Disorders of Speech. He was a pioneer in many domains of medicine. It is hardly known that he was the first one to describe osteomyelitis, to attempt the construction of an ophthalmoscope, or to develop gastro- and esophagoscopy, to establish the relation between anemia and convulsions; the first one to point out the importance of coryza for the suckling baby, to describe bulbar paralysis, to describe and analyze diabetic coma, to introduce the method of thoracocentesis, the first one to diagnose *intra vitam* embolism of the mesenteric artery and to describe the diagnostic significance of the pulsus paradoxus. There are a good many more important contributions to general medicine which we cannot mention here. But we have yet to speak of his great service to gastro-enterology. He was the first to employ a stomach tube, and by this step he undoubtedly gave the first and most important impulse to the

modern development of our knowledge of the diseases of the stomach. His articles on the treatment of the dilatation of the stomach and on the peristaltic unrest of the stomach are classical contributions to our subject. Many important additions to our knowledge have been published by his assistants and pupils under his direct influence and active cooperation. Of those which interest us here I shall mention only the following few: On the presence and absence of hydrochloric acid in gastric juice by Van den Velden; the classical prize essay by Poensgen on the motor activities of the human stomach; on the treatment of the ileus by washing the stomach, by Arnold Cahn; on the acids of the healthy and diseased stomach by Cahn and von Mehring. Kussmaul's last contributions to gastro-enterology were made from his retreat in Heidelberg while already over seventy; they are practical and of permanent value, namely, the treatment of gastric ulcer by large doses of bismuth, and the treatment of chronic constipation by enemata with large quantities of oil.

When already over seventy-five years he wrote his celebrated autobiography, Juvenile Recollections of an Old Practitioner, an inspiring, fascinating little book. Old age had not curtailed the usefulness of that great and lovable man. I shall finish with the statement that to my knowledge Kussmaul never attempted to write a textbook.

It might not be unprofitable to speak of Kussmaul as a practitioner and clinician. All his assistants and pupils could not praise enough his kindness and devotion to his patients. While he was an enthusiastic investigator, he never permitted the study of a case in a manner which could bring harm or discomfort to the patient. The patient was always first and science second, a high principle which should be burned into the minds of our generation of overproductive specialists. Kussmaul told his students he wished them to pass through a serious disease, it would help them to become good physicians, they would then understand what care, kindness and tenderness meant to a suffering patient. Kussmaul himself learned this lesson during his own sick-

ness and did not forget it for half a century. He insisted that his students should learn the particulars of nursing, the making of a bed, the handling of a patient, the preparation of his food, etc. Above all he laid great stress upon the moral and mental influence of the physician upon his patient. He believed in making the patient at least hopeful and cheerful even if he could not be made well. Kussmaul was a great and good physician even if he did not believe in the more godly though less humane doctrine of Truth in Medicine. May the life of this great man be an inspiring example to the younger generation in this country.

Samuel Fenwick died December 11, 1902, at the age of eighty-one years, of heart failure, due to atheroma of the aortic valves. He was born in 1821 in Northumberland, England, of a family which played a conspicuous part in that country for seven centuries. He was compelled to study medicine on account of pecuniary losses which his father had sustained. At the age of fourteen he was apprenticed to the Royal Infirmary of Newcastle-on-Tyne, from which, after seven years, he graduated as M.R.C.S., England. In 1846 he obtained the title of M.D. from the University of St. Andrews. While he was still in the twenties he undertook original researches upon the process of digestion and absorption in lower animals, wrote a book on the chronic diseases of the throat and was appointed lecturer on pathological anatomy at the Durham School of Medicine. He moved later to Newcastle where he acquired a consulting practice said to have extended over the whole of the north of England. In 1859 he was made honorary M.D. by the School of Medicine of Durham. In 1862 he moved to London where he acquired an extensive consulting practice which he carried on uninterruptedly to the very last days of his life. His private practice seems to have been confined essentially to the diseases of the stomach. In London Hospital, however, where he went through all grades until he became senior physician, he taught many branches of medicine, beginning with physiology. Fenwick was a prolific writer on many subjects. He wrote several

text-books which passed through a number of editions. In the later years of his life, his writings were mostly on subjects of gastric diseases. He was probably among the first to write a book on the diseases of the stomach. It was in 1865 and the title reads: "Morbid States of the Stomach and Duodenum." The ninth edition of that book was revised by him fourteen days before his death. He was a man of dignified reserve. He led a retired home life, which was due, it is stated, to a very delicate stomach, a condition which might have been also the reason for his paying special attention to the morbid conditions of that organ.

Franz Riegel died August 26, 1904, at the age of sixty-one, of a malignant neoplasm of the lungs. He was born February 9, 1843, at Brückenau, and was the son of a physician. He studied medicine at Würzburg and after graduating there with highest honors, took a post-graduate course in Vienna under Oppolzer and Stricker. Returning to Würzburg he became assistant first to Bamberger and later to Gerhardt, both celebrated clinicians of their time. In 1871 Riegel became privat-docent at the university, where he lectured on experimental pathology. In 1874 he was appointed director of the City Hospital in Köln, whence he was called (1879) to the University of Giessen to the chair of medicine, which was first filled before him by such celebrated clinicians as Niemeyer and Seitz. The twenty-fifth anniversary of this appointment was celebrated last May by his many pupils, friends and admirers, his assistants and pupils dedicating to him a valuable and extensive Festschrift. He then commenced to show the distressing symptoms of his vicious malady, which, after a few months of suffering, took him away from us in the midst of his active, useful life.

Riegel was a very earnest character, a savant who knew no leisure and no amusement, but hard work and earnest studies which to the last days of his fruitful life he devoted to the science and practice of medicine. His publications are numerous and all very valuable. Differing from most of the distinguished clinicians of his time for whom pathological anatomy was the es-

sential factor of their scientific productivity, all of Riegel's works bear a physiological stamp, his interest centered in functional problems of life, and many of his clinical observations were supplemented by thorough experimental studies. His biographers say that the foundation for this direction of mental activity was laid in Vienna by his contact with Stricker. While I do not doubt that Stricker exercised a certain influence upon the future scientific direction of Riegel, I personally believe that the inclination for the study of functional rather than morphological problems is an inherent one with the individual. It was, so to say, congenital with Riegel. The subject of his thesis, which was written before he met Stricker, was already of a physiological character. It dealt with the respiratory movements in health and disease. Later he returned again and again to the studies of the respiratory organs, to stethoscopy, to the nature of asthma and its treatment, etc. What a fate to die of a malignant disease of an organ to the study of which he had paid so much attention! Undoubtedly he must have made his own diagnosis. Other extensive studies were devoted to the vasomotor influences, to the arterial pulse, to the venous pulse, to the contractions of the heart, to the conditions of the heart and blood pressure in nephritis, to the effects of jaborandi, caffeine, atropine and other drugs. A good part of his work, at least during the last two decades of his life, was devoted to the diseases of the stomach in which field he undoubtedly occupied the first rank. It would nearly be writing a detailed history of the present state of knowledge of gastric disease, were I to attempt to count up the work which Riegel and his able assistants have done in this field. By the present almost exclusive use of a test breakfast, it is practically forgotten that the importance of this method lies in the test meal and that this was first used and introduced by Riegel in 1879. The last work coming from the pen of Riegel was devoted to a discussion of the distinction between hyperacidity and hypersecretion. It is of psychological moment to know that Riegel himself often suffered from gastralgia accompanied by hypersecretion.

By the undertaking of Nothnagel to publish classical monographs on all chapters of medicine by leading clinicians, Riegel was induced to treat of the diseases of the stomach; it resulted in the production of a masterpiece. In the last months of his life he was engaged in the preparation of a second edition of this classical book. It was one of his great worries that he was unable to finish the revision of it. Let us hope that one of his many able assistants will be able to complete it in the spirit of the dead master.

[Reprinted from THE MEDICAL NEWS, May 20, 1905.]

RECENT ADVANCES IN THE KNOWLEDGE OF THE MOVEMENTS AND INNERVATION OF THE ALIMENTARY CANAL.*

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THE mechanical factors in digestion perform three chief functions, the movement of the food through the alimentary canal, the mixture of the food with the digestive juices, and the exposure of the food to the absorbing mucous membrane. The progressive peristaltic wave is the most general activity by which these functions are effected. But the progression of the food through the canal is not uniform. Where digestive juices are lacking and absorption does not occur, as in the esophagus, the food is moved rapidly. On the other hand, where digestion and absorption can take place, rapid progression is prevented by sphincters; and the recurring peristaltic waves passing over the food toward closed sphincters serve to mix the food with the digestive juices, as in the stomach, or expose the food to the absorbing mucosa, as in the ascending colon. In the long course of the small intestine, with no sphincters present to oppose peristalsis, peristaltic activity is infrequent, and the mixing and exposing functions are carried on by a special method,—the rhythmic contraction of the circular fibers. These rapidly repeated contractions knead the intestinal contents, without causing any considerable progression.

From the foregoing general considerations it is evident that different portions of the alimentary canal are characterized by particular adaptations of the general method of forwarding, mixing and exposing the food. In this paper the mechanical activities of the different portions of the canal—esophagus. [illegible]ch, small intestine, and large in-

* Present[illegible]eting of the American Gastro-enterological Assoc[illegible]rk, April 24, 1905.

testine, with the sphincters—will be separately treated. As my time is limited I am compelled to neglect details and describe only the more important new accessions to our knowledge.

THE MOVEMENTS OF THE ALIMENTARY CANAL.

The Esophagus.—As the word implies, the esophagus is merely a food carrier, serving to transmit nutriment rapidly from one digestive region to another. The name of your pesident (Dr. Meltzer) is associated with the first demonstration that the swallowed material may be forced through the esophagus by a pressure in the mouth so great that the esophagus acts not to carry but to direct the discharge from the pharynx.[1] From these and subsequent observations[2] the conclusion was drawn that a fluid or semisolid morsel is thrown down the entire length of the esophagus by the contraction of the mylohyoid muscles, and that the activity of the esophagus is merely supplementary, to gather the particles left along the way, or to push down an occasional dry bolus. The possible influence of the method—a tube in the esophagus—in producing the results secured by Kronecker and Meltzer led to the repetition of the observations on man and lower animals in the Harvard Physiological Laboratory.[3] The observations were made by means of the Roentgen rays, and the movement of the food was not affected by the presence of apparatus. The results entirely confirmed those of Kronecker and Meltzer so far as liquids are concerned, but they failed to show any shooting of semisolid food or even thick fluids along the canal. In all the animals investigated (fowl, cat, dog, horse and man) the progression of semisolid and firm particles toward the stomach was accomplished by contraction of the esophagus.

According to Kronecker and Meltzer[2] the human esophagus may be divided into three parts; a cervical part 6 cm. long, a middle part 10 cm. long, and the lowest part about 6 cm. in length. These three parts contract in succession, 1.2, 3, and 6 seconds, respectively, after deglutition be-

gins, but each part, according to Meltzer, contracts as a unit, simultaneously throughout its length. The rate of contraction is different in the different parts. The human esophagus therefore would undergo three successive sectional contractions, not peristaltic in nature.†

Schreiber[4] has studied the contractions of the human esophagus with the method used by Kronecker and Meltzer, and finds no separation of the canal into three parts, each with its own time for contraction. Instead, his curves reveal the existence of a constriction registered gradually later as the recording apparatus is placed gradually deeper in the esophagus. This moving constriction can be explained only as a peristaltic wave. The wave at a given point lasts in the neck region about 3.5 seconds, and from 5 to 9 seconds in the thoracic region. Schreiber's conclusion that the wave moves rapidly in the cervical portion and slower in the thoracic portion, with no pause between the two, confirms the observations of Moser and myself on lower animals. We noted[3] in the dog and cat that food was pushed onward rapidly in the upper part of the esophagus, and considerably less rapidly from the top of thorax or the level of the heart to the stomach. If the esophagus contracts in sections, with an interval of two or three seconds between the contraction of adjoining sections, a checking of the progress of the bolus should be expected at the change from the rapid to the slower rate. But no interruption of the onward movement was observed in the dog, and only occasionally was it seen in the cat—there was merely a gradual slackening of the speed as the bolus reached the lower esophagus.

The Cardia.—It is generally stated that normally the cardiac sphincter is in a state of tonic contraction. As long ago as 1860, however, Basslinger[5] described a rhythmic alternation of contraction and relaxation of the rabbit's cardia.

† Dr. Meltzer states (New York Medical Journal, 1894, LIX, p. 391), before generalizing, that these results are true, at least, of his esophagus.

In observations on the cat (still unpublished) I have seen rhythmic relaxations of the cardia, so that fluid food streams from the stomach into the esophagus even above the level of the heart, then is pressed into the stomach again by a peristaltic wave, only to be released a moment later to pour into the esophagus anew. I have watched this activity recurring at intervals of one or two minutes for more than a half hour. It usually begins soon after fluid food is given; within a varying period the intervals between discharges gradually lengthen until the action entirely ceases and the cardia seems tightly closed. During this ebb and flow of food in the esophagus the animal evinces no signs of distress, and if allowed her freedom, does not vomit. On re-examination the movements are again seen. The regular appearance of these phenomena in different animals makes me think that they must be normal. It may be that relaxations of the cardia of this character are associated with eructations of gas and stomach contents in cases of gastric fermentation.

Kronecker and Meltzer have maintained that even when food is squirted down the esophagus, it normally rests above the cardia until pressed through into the stomach by esophageal contraction. But if several swallowings follow one upon another, the cardiac sphincter becomes relaxed, so that the movement of the food to the stomach meets no resistance.[6]

In rabbits, after a peristaltic wave had pushed the food from the esophagus into the stomach, Meltzer observed[6] a strong contraction of the sphincter, immediately followed by a deep diving of the entire cardia into the stomach, as if pressing down the contents of a filled bag.

The functions of the cardia therefore may be to keep the passage into the stomach free from an accumulation of the swallowed food, and, so long as the sphincter remains contracted, to prevent a regurgitation of gastric contents.

The Stomach.—The stomach is a region of storage for the food, and also a region in which carbohydrate and proteid digestion may occur.

These functions of the stomach are performed in different parts of the organ.

Undoubtedly the most important feature of the newer physiology of the mechanics of the stomach is the knowledge that it consists of two parts physiologically distinct. The larger left part of the stomach is the cardiac portion, the right is the pyloric portion. The pyloric portion is characterized during digestion by the continuous passage of peristaltic waves over its surface to the pylorus.[7] The cardiac portion is without peristalsis, but as the food is pressed from the pyloric portion into the intestine, the muscles of the fundus bv tonic contraction squeeze the contents into the more active division, as into a hopper.[8]

The rate of gastric peristalsis varies with different animals. In the cat four to six waves pass a given point in a minute; in the dog, between four and five.[9] Moritz[7] states that in the human being the rate is about three per minute. I have confirmed his statement by means of auscultation. When food containing air is eaten, and the subject lies on his left side, a sound can be heard over the pylorus recurring rhythmically at intervals of about 19 or 20 seconds.

The efficiency of peristalsis in mixing the food with the gastric secretions depends upon the contraction of the pyloric sphincter. So long as the sphincter holds, each constriction-ring coursing from the middle to the end of the stomach presses the food into a blind pouch; the food, unable to escape through the pyloric opening, has as its only outlet the opening in the advancing ring. This is an admirable device for bringing the food under the influence of the glandular secretions of the pyloric region. For, as a constriction occurs, the secreting surface enclosed by the narrowed muscular ring is pressed close around the food within the ring. As the constriction advances it continually presses inward fresh glandular tissue, and furthermore, as the constriction advances, a thin stream of food is continuously forced back through the ring and thus past the mouths of the glands—a food stream sweeping in

one direction past an ever-refreshed mucous surface moving in the opposite direction. Thus is the food repeatedly exposed to the secreting surface of the pyloric end and thoroughly mixed with the gastric juice.

The larger cardiac end of the stomach is the reservoir for the food. As peristaltic waves are absent, the food remains unmixed with the gastic juice and the inner parts may retain their original reaction for even two hours. In 1898 I pointed out that in this larger part of the stomach salivary digestion may readily take place without interference by the acid gastric secretion. In 1903 Mr. Day and I[10] proved that when carbohydrate food is mixed with saliva the cardiac portion contains at the end of an hour about 80 per cent. more sugar in unit volume than the pyloric portion. These observations have been confirmed by Grützner,[11] who has also shown that when new food enters a partially full stomach, it passes into the midst of the cardiac mass and displaces outward the food already there. The possibility of salivary digestion in the stomach is thereby assured.

The value of the stomach as a place for holding the bulk of the food and serving it out so gradually that the intestine is not overwhelmed during its digestive and absorptive processes, was pointed out by Moritz.[12] The advantages thus secured to the intestine may be claimed also for the stomach itself, for the active portion is the pyloric portion, and the fundus serves as its resvoir—an active reservoir pressing out its contents a little at a time, as the pyloric peristaltic mechanism is ready to receive them.

The Pylorus.—Differing views have been set forth as to the manner in which the pylorus opens and permits the exit of the food. Some investigators[13] have declared that the sphincter relaxed only at the end of several hours to allow the stomach to empty. Observations under more natural conditions, with the stomach-tube[14] and with the X-rays,[8] show that the stomach is not emptied at once at the end of gastric digestion, but pro-

gressively during the period. There is thus an intermittent closure of the pylorus. The channel is usually closed, yet occasionally it opens; and when it opens, the peristaltic waves, usually engaged in churning the food, now serve to propel it into the intestine.

The factors affecting the pylorus are as yet but little understood. It is known that the discharge of food from the stomach is delayed by the presence of acid[15] or fat[16] in the duodenum. Hard particles brought to the sphincter by gastric peristalsis also prolong the periods of contraction.[8] Under these circumstances the hard particles are repeatedly swept up to the pylorus, only to be repeatedly shot back through the advancing ring of constriction. By this continual rubbing the waves of peristalsis serve to break up the more solid pieces of food, a function performed by the teeth with manifestly greater efficiency. It is possible that the prevalence of pathological conditions in the pyloric end of the stomach, rather than in the cardiac end, is due to injuries which the greater activity of the pyloric end may bring upon itself.

What agency causes the pyloric sphincter to relax? It seems probable that the signal for relaxation is the presence of free hydrochloric acid on the stomach side of the pylorus. Penzoldt[17] noted that foods delaying the appearance of free hydrochloric acid remained longest in the stomach. His observations, however, did not decide which of the factors, gastric peristalsis or the pylorus, caused the retardation of the food. In the Harvard Physiological Laboratory last year it was proved[18] that when carbohydrates, proteids and fats of the same consistency are separtely fed in equal amounts, they do not leave the stomach at the same rate. Fats remain long in the stomach; the discharge begins slowly and continues about as rapidly as the fats are absorbed or passed into the large intestine. Carbohydrates begin to leave the stomach soon after their ingestion (within ten minutes); they pass out rapidly and at the end of two hours reach a maximum amount

in the small intestine,—almost twice the maximum for proteids and two and one-half times the maximum for fats,—both of which maxima are reached only at the end of four hours. Proteids frequently do not leave the stomach at all during the first half hour and occasionally not for an hour. After two hours they accumulate in the small intestine to a degree only slightly greater than that reached by carbohydrates an hour and a half earlier. The departure of proteids from the stomach is much later than that of carbohydrates and slower than either carbohydrates or fats. There is evidently a remarkable mechanism at the pylorus which permits carbohydrates, not digested by the gastric juice, to pass quickly into the intestine where they are digested, and retains the proteids, digested in the stomach, there to undergo digestion.

If the presence of free acid on the stomach side of the pylorus stimulates the relaxation of the sphincter, this mechanism is largely explained. The theory of the action of the pylorus which I suggest is as follows:[19] Free acid in the stomach opens the pylorus and initiates the chemical control of the sphincter. The opening of the pylorus permits the exit of a portion of the acid chyme. We have seen that acid in the duodenum keeps the pylorus closed; but acid in the duodenum also stimulates the flow of the alkaline pancreatic secretion.[20] No inorganic acid is normally present beyond the first few inches of the small intestine.[21] The acid therefore is here neutralized. As the neutralizing proceeds, the stimulus closing the pylorus is weakened, until the acid in the stomach again opens the sphincter. Again the acid food passes out and the acid closes the sphincter to further passage until the duodenal changes finish their slower course.

Thus, automatically, carbohydrates would go early and quickly into the intestine to meet their proper ferment; thus, automatically, proteids, postponing the presence of free hydrochloric acid by uniting with it, would be retained in the stomach to suffer alternation by the gastric juice and

only after such alteration be permited to go on. Thus, automatically, the intestine would be saved from overwhelming discharges from the gastric reservoir; and by this same automatic mechanism the gastric secretion, harmful to the action of intestinal ferments, is rendered innocuous because of its admission, little by little, into the duodenum.

The Small Intestine.—The small intestine is not only a region of digestion but also a region of absorption. Throughout its length there are no sphincters. Peristalsis cannot act therefore to mix the food with the secretions or expose the digested food to the absorbing wall. The admirable process by which these functions are performed, I have called " rhythmic segmentation."[22] In the small intestine there are then two mechanical activities, rhythmic segmentation of the food, the most common activity, and peristalsis. The rhythmic movements of the small intestine have been variously described as shortenings and narrowings of the gut, rhythmically repeated at nearly the same intestinal circumference; as alternating to-and-fro movements of the long axis without changes in the lumen; as local or extensive periodic contractions and relaxations mainly of the circular musculature; and as waves involving both muscular coats of the intestine and traveling from above downward at a rapid rate.[22] Ludwig gave these rhythmic movements the rather undescriptive name " Pendelbewegung," or pendulum movement. Some of the rhythmic movements above mentioned are undoubtedly abnormal and due to the method of observation employed. The effect of the movements in normal cases is to mix the food and expose it to the mucosa without advancing it appreciatively along the canal.

In this process of rhythmic segmentation a mass of food which has been lying at rest is separated into a number of equal segments by constrictions of the circular muscles occurring at intervals along its length.[22] Scarcely have the constrictions formed when similar constrictions begin to appear about the middle of each segment. As

these new constrictions deepen, the first constrictions begin to relax. Finally, when the new constrictions have completely divided the segments, the first constrictions have entirely relaxed and the neighboring halves of the divided segments unite to form new segments in the region of the first constriction. These new segments are again divided by circular constrictions about their middle, and neighboring halves of these divided segments unite to make a third series, and so on. This process of rhythmic segmentation of the food may go on for several minutes in the duodenum so that the food, coming from the stomach, becomes thoroughly mixed with the outpouring secretions of the liver and pancreas. It is usual for the process in the duodenum to be brought to an abrupt end by active peristalsis. In other parts of the small intestine rhythmic segmentation of the food may continue for half or three quarters of an hour without moving perceptibly along the canal. In the cat the segmentation may occur about thirty times in a minute. The food may thus be divided and redivided over and over again a thousand times or more with no advance. This operation has been observed in the dog at the rate of from eighteen to twenty-two segmentations per minute. I have never watched the movements of the small intestine in man, but I have been able with the stethoscope to hear in the lower abdomen rhythmic sounds repeated with remarkable regularity at intervals of seven or eight seconds. It seems to me probable that these sounds are associated with movements of rhythmic segmentation, for I have heard similar sounds attending segmentation in the cat. If this is true the rate of segmentation in the human being would be seven or eight times per minute. The process of rhythmic segmentation thoroughly mixes the food with the digestive secretions of the liver and pancreas and of the intestine itself, and thoroughly exposes the digested food to the absorbing walls, for, with each constriction, the absorbing mucous membrane is plunged directly into the midst of the little masses of food. This

remarkable activity must also affect the flow of blood and lymph, since Mall[23] has shown that contraction of the intestinal wall forces venous blood from the submucous plexus into the radicles of the superior mesenteric vein and has noted that it must similarly affect the flow of the lymph.

The progression of the food through the small intestine is possibly caused to a very slight extent by the segmenting process, but it is typically performed from time to time during digestion by the well-known peristaltic wave, which has so long been regarded as the chief activity of the intestinal muscles.

The Ileocolic Sphincter.—In 1902 Katz and Winkler,[24] and in 1904 Elliott[25] demonstrated that at the junction of the small and large intestines there is, at least in the dog, cat and rabbit, a distinct sphincter under nervous control, and not a mechanical valve. This sphincter is normally in a state of tonic contraction and serves to keep separated the contents of the small from those of the large intestine. There is some anatomical and histological evidence that also in man the term "ileocecal valve" should be replaced by "ileocolic sphincter," but physiological evidence of the presence of this sphincter in human beings has not yet been produced.

The Large Intestine.—When the large intestine is full the material in the distal portion is usually composed of rather hard lumps, while that in the proximal portion is soft and of uniform consistency. The condition of the contents in these two regions indicates a division of the large intestine into two parts, and the mechanical activities of these two parts correspond to the differentiation. In the distal portion the material is slowly advanced by rings of tonic constriction. In the proximal portion the common movements are waves of constriction running *backward* toward the cecum.

The antiperistalsis of the proximal portion of the large intestine was incidentally observed in the cat by Jacobj[26] during a research on colchicum poisoning. In 1902 this activity of the large

intestine was established as the normal activity in the undisturbed animal by observations with the Roentgen rays in the Harvard Physiological Laboratory.[22] In 1904 Elliott and Barclay-Smith[27] confirmed these observations on the cat and found that in the rat, guinea-pig, rabbit, and occasionally in the hedgehog and ferret, antiperistalsis may be observed in the proximal colon. They also point out that the contents of the human cecum and ascending colon are of uniform soft consistency, whereas those of the transverse and descending colon are generally firm. As these distinctions correspond to those observed in animals in which antiperistalsis in the proximal large intestine has been seen, it seems highly probable that the human cecum and ascending colon are likewise characterized by this activity.

The antiperistalsis in the large intestine makes reasonable the presence of a sphincter or valvular structure at the point where the small intestine enters the large, for in so far as the sphincter is competent to restrain the food in the large intestine from returning into the small, the antiperistaltic waves have the same effect as the peristaltic waves in the stomach, pushing the food against the closed pylorus. When a constriction occurs some of the mucous surface of the colon becomes enclosed by the narrowed muscular ring, and, as this constriction passes on, fresh areas of this surface are pressed in around the narrow orifice, while a thin stream of food is passing by it in the opposite direction. The waves recur about five times a minute in the cat, and the period of activity, which lasts about five minutes, is usually repeated every fifteen or twenty minutes. During each normal period of antiperistalsis twenty-five waves or more must pass over the food in the cecum and proximal colon in this churning manner. The result must be again a thorough mixing of the contents with the digestive secretions brought from the small intestine, and an exposure of the digested food to the absorbing wall.

In 1894 Grützner declared that he had found material, injected into the rectum, carried back

into the small intestine even as far as the stomach. Since then some observers have confirmed, others have doubted Grützner's results. The presence of antiperistalsis in the colon led me to believe there existed an activity which would fully account for Grützner's findings. Accordingly I injected into the large intestine enemata of different amounts and of different consistency. In every instance antiperistaltic waves were started by the injected mass and the material was therby carried in considerable amount to the cecum. Never in my experience have small or mushy enemata been carried back through the ileocolic sphincter. But when large injections were given, even of the consistency of thick cream, *i.e.*, amounts nearly filling the colon and cecum, the antiperistaltic waves may carry the food from the large intestine into the ileum. Radiographs taken at different intervals after the injection show that the antiperistaltic waves as time passes may almost completely empty the proximal colon, and that the small intestine gradually receives more and more food. After a short time typical segmenting movements can be seen in the loops.[22] Evidently, so far as mechanical activities are concerned, the small intestine is bipolar: food entering at either end is worked over with the same mixing and absorbing activites.

With each new accession of food in the large intestine a strong tonic constriction of the cecum and proximal colon occurs, which presses onward into the distal colon some of the contents of these parts. Antiperistaltic waves follow at once the tonic contraction, so that much of the food which has been pressed onward is returned toward the cecum. With the repetition of this process, however, more and more material accumulates in the distal colon until finally a persistent ring of contraction separates this material from the region of antiperistalsis; as still more food appears in the large intestine this ring moves slowly onward toward the rectum, pressing the mass before it, and is followed by other similar rings carrying onward similar masses by very slow peristalsis.

The masses of waste material thus advanced accumulate in the lower part of the distal colon and are ultimately passed from the body in the act of defecation.

In the cat defecation is preceded by the disappearance of the slowly moving peristaltic rings in the lower part of the distal colon and the appearance of a strong, broad constriction near the beginning of this region. The band of constriction is then drawn downward by a shortening of the longitudinal muscles. Thereupon the constriction, which divides the lumen, moves slowly along and, with the aid of the voluntary abdominal muscles, pushes the separated mass through the relaxed sphincters out of the canal.[22] Elliott and Barclay-Smith[27] observed in all the animals they studied a similar powerful constriction of the tube occurring at a definite point some distance up the colon and spreading thence downward so as to empty the distal segment, and in many animals there was also a contraction of the longitudinal muscle which, fixed in the perineal region, draws the colon down into the pelvis and thereby aids the constriction that performs evacuation.

THE INNERVATION OF THE ALIMENTARY CANAL.

In the foregoing review of the motor activities of the alimentary canal the main factors are shown to be the moving peristaltic wave and the stationary rhythmic contraction of circular muscle. To what extent are these movements independent of the central nervous system, and in what manner are they affected by their connection with that system? Before attempting to answer this question, it will be necessary to know the nervous relations of the alimentary canal.

Throughout the canal there lies, between the inner circular and the outer longitudinal fibers, a nerve net or plexus,[28] of primitive structure, characteristic of the walls of viscera and of simple animals such as the cœlenterates. This nerve net (Auerbach's plexus) provides a local nervous mechanism.

Besides a plexus system the alimentary tract

possesses intimate connections with the spinal cord and brain. The vagus nerve is distributed to esophagus, stomach, and small intestine; and pelvic visceral nerves, to the large intestine. Moreover the sympathetic nervous system sends fibers to the entire canal from the cardia to the end of the colon.

The Local Nervous Mechanism.—The simplest form of activity in the alimentary canal is the local contraction of the circular fibers, the process of rhythmic segmentation. Bayliss and Starling found that in the dog with abdomen opened this movement consisted of constrictions repeatedly recurring at the same spot and traveling along the gut from above downward. When nicotine was injected into the blood vessels or cocaine was painted on the intestine, they found that the true peristaltic wave was abolished, but that rhythmic contractions continued.[29] The rhythmic contractions now traveled in either direction, instead of downward only. As cocaine and nicotine, in the amounts used, paralyze nerve cells, the rhythmic contractions were regarded as of muscular origin, and the transmission of the contraction a muscular transmission. Last year Magnus[30] succeeded in recording graphically rhythmic contractions of circular and longitudinal muscles of the intestine removed from the body and kept alive in warm salt solution. The contractions persisted after the removal of the mucosa and Meissner's plexus; but after separation of the circular and longitudinal muscular coats, the circular coat would respond to stimulation, but only the longitudinal coat registered the spontaneous rhythm. Examination proved that Auerbach's plexus was adherent to the longitudinal coat. Magnus concluded therefore that the rhythmic movements are of nervous origin. The question as to the neurogenic or myogenic origin of the rhythmic movements of the small intestine is therefore an open one.

The peristaltic activity of the alimentary tract shows different degrees of dependence on nervous influence. Elliott and Barclay-Smith[27] state

that antiperistaltic waves of the large intestine are not abolished by nicotine. They class these waves with the rhythmic movements of the small intestine, as of myogenic origin.

The true perstalsis of the small intestine, according to Bayliss and Starling,[29] and likewise of the large intestine, according to Elliott and Barclay-Smith,[27] is a coordinated movement, dependent on a reflex in the wall of the canal. The movement continues after separation from the central nervous system, but is under local nervous control since it is destroyed by cocaine or nicotine. And, as Magnus has proved,[30] the seat of the reflex is in Auerbach's plexus. It is probable that the same reflex governs the stomach movements, for gastric peristalsis continues after the stomach is removed from the body.[31]

The local reflex causes the canal to respond in a uniform manner to stimulation. The uniformity of the response has led Bayliss and Starling to formulate a *law of peristalsis.*—"Local stimulation of the gut produces excitation above and inhibition below the excited spot." So long as the reflex mechanism is intact in the stomach, the small intestine, and the distal colon, antiperistalsis cannot occur—a conclusion which Mall's[32] observations on reversed loops of intestine fully support.

The peristalsis of the esophagus is singularly closely related to the central nervous system. For example, Mosso[33] proved that cutting across, or even extirpating a piece of esophagus did not stop the peristaltic wave, if connections with the central nervous system remained intact,—the wave, started above the cut, reappears below. Yet, as Meltzer[34] has shown, there is also in the esophagus a local reflex like that in the remainder of the alimentary canal; and if anesthesia is so deep that central nervous influences are eliminated, at least in part, from control of the peristaltic wave, the wave will move, but may be stopped by cutting or ligating the esophagus. On the other hand, it has been known since Reid's observations in 1839[35] that severance of the vagus

nerves in the neck deeply disturbs the activity of this region, so that food accumulates in the esophagus and may remain there for hours without being advanced. I have determined that the delay is not due to spasm of the cardia, but to a paralysis of the esophagus.[36] It seems probable that the peristaltic contractions of the esophagus, to be efficient, must be supported by nervous influences from the outside. In this respect the esophagus is different from the remainder of the alimentary canal.

The Nervous Control of the Sphincters.—Whether the sphincters are controlled by the local nervous reflex active elsewhere in the alimentary tract is not yet experimentally settled. So far as the pylorus is concerned, cutting either the vagus nerves or the splanchnics does not destroy the mechanism maintaining the difference between the discharge of proteids and carbohydrates from the stomach.[36] Whatever the local control may be, the sphincters are certainly markedly affected by their connection with the central nervous system. It must be confessed, however, that our knowledge of the extrinsic innervation of the alimentary canal is still in an unsatisfactory state. The many contradictory observations cannot be set down here; I shall endeavor to present only the later and best-founded conclusions.

The observations of Kronecker and Meltzer,[2] and Openchowski[37] have proved that the vagus nerve carries to the cardia inhibitory impulses. Recently May[38] has declared that although the first effect of vagus stimulation is inhibitory, there is later a slow augmentation of the contraction. Langley[39] was able to separate inhibitory and augmentor impulses in the vagus with drugs; after curare, vagus stimulation caused contraction of the cardia, and after atropine, the same stimulation caused inhibition. Stimulation of sympathetic fibers, according to Openchowski,[37] brings about increased contraction of the sphincter.

At the pylorus the usual result of vagus stimulation is contraction,[37] although relaxation is occasionally observed. May[38] states that stimu-

lating the vagus has the same effect on the pylorus that it has on the cardia,—first an inhibition, followed shortly by an augmentation of contraction. Impulses from the splanchnics, so Openchowski states,[37] are chiefly inhibitory of pyloric contraction in the dog, but in the rabbit they are motor.

The ileocolic sphincter is not affected by the vagus, but stimulation of sympathetic nerves causes the sphincter to contract, and at the same time inhibits the circular muscle of adjoining parts of the ileum and colon.[25]

The Extrinsic Innervation of the Stomach and Intestines.—The vagus nerves contain motor as well as inhibitory fibers for the stomach. According to May[38] the stimulation of the vagus results in the same changes that have been described for the cardiac and pyloric sphincters; there is first an inhibition of the peristalsis of the pyloric end of the stomach and a lessening of muscular tone in the cardiac end, and this is followed by a much more vigorous peristalsis and, in the cardiac end, by an increased tone.

Most observers attribute to the sympathetic fibers of the stomach inhibitory functions alone; but Morat[40] has noted the opposite effect, and Openchowski[37] states that in the rabbit the sympathetic has chiefly a motor influence. May[38] fails to find any effect, whatever following stimulation of the splanchnic nerves.

There is a greater unanimity of opinion regarding the motor innervation of the small intestine,—almost all observers ascribe to the vagi an excitatory influence. Bayliss and Starling[29] found that invariably stimulation of these nerves produced, after only a momentary inhibition, a marked and prolonged augmentation of the contractions of the circular muscles. On the other hand, although the very careful work of Bayliss and Starling[41] has disclosed only inhibitory action of the splanchnic fibers, many other observers have recorded a motor effect as well.[42]

The results secured by Bayliss and Starling, in their study of the innervation of the large intestine,[43] are quite in harmony with what they found

true of the small intestine,—the pelvic visceral nerves arising like the vagus from the central nervous system, are augmentor nerves, whereas the supply from the sympathetic system is purely inhibitory in its action. Elliott and Barclay-Smith[27] have confirmed the observations of Bayliss and Starling, but find that the pelvic visceral nerves are distributed only to the distal part of the colon. The region of antiperistalsis does not, therefore, receive motor impulses from the pelvic nerves.

Inhibition of the Movements of the Alimentary Canal During Emotions.—Observations on the cat, made in the Harvard Physiological Laboratory in 1898, showed that gastric peristalsis is stopped whenever the animal manifests signs of rage, distress, or even anxiety.[8] With the similarity between the extrinsic innervation of the stomach and that of the intestinal tract, it was of interest to note that emotional states have the same effect on movements of the intestines that they have on movements of the stomach. Any signs of emotional disturbance, even restlessness and continued mewing, which indicate, perhaps, uneasiness and discomfort, were accompanied by total cessation of segmenting movements in the small intestine and antiperistalsis in the proximal colon, as well as complete quiescence of the gastric mechanism. Such signs of anxiety I have seen accompanied by entire inactivity of every part of the alimentary canal during more than an hour of continuous watching.[22] My observations on cats have been fully verified by Lommel,[44] who worked with dogs. Evidently chemical factors of digestion can have little importance, if fears and worry prevent the delivery of the food to regions of active digestion and absorption.

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DISCUSSION.

DR. FREDERIC S. LEE, of New York City: Professor Cannon has modestly refrained from emphasizing the part which he has taken in the work that he has detailed, and I feel that it is almost idle to invade a field in which he has labored so ingeniously and so successfully. In considering his paper there is but one point which I will mention.

There has long been strife among physiologists as to whether the beats of the heart are of muscular or of nervous origin. The old idea that they were primarily nervous was supplanted by the muscular theory, but recent investigations seem to show that, in some species of animals at least, the heart beats as a result of impulses coming to the muscle cells from nerve tissue. It is quite possible therefore that we shall be forced back again to the neurogenic theory of heart action. The same problem arises in connection with other muscular structures. Apathy and Bethe claim that the functional unit of the nervous system is the fibril, not the neuron with its processes. They have demonstrated in many tissues a rich nervous network composed of fibrillae which are smaller than axis cylinders: and Bethe especially is inclined to ascribe all cases of rythmic contraction primarily to the action of nervous substance. As to the movements of the alimentary canal, the arguments that are usually quoted in support of their muscular origin seem to me to be not altogether conclusive, and the work of Apathy and Bethe on nerve fibrils, together with that of Carlson on the origin of the heart beat, makes us question whether there will not yet be demonstrated in the walls of the alimentary canal a nervous network, much more minute than any that has been formed which is connected with the muscle layers and is directly responsible for peristalsis.

DR. MAX EINHORN, New York City: I feel very thankful to Dr. Cannon for the opportunity to hear his views on this important subject, and fear I cannot add very much to what he has said.

Dr. Cannon said it seemed probable that the signal for the relaxation of the pyloric sphincter was the presence of free hydrochloric acid on the stomach side of the pylorus. I am a little doubtful about accepting that explanation, because while this would be a satisfactory theory under normal physiological conditions, it does not explain the relaxation of the sphincter in cases, for example, of achylia gastrica, where there is no acid in the stomach, and still the food finds an exit into the intestines. There must be other factors that play a part in the relaxation of the pyloric sphincter.

DR. JOHN C. HEMMETER, of Baltimore: In looking through the œsophagoscope I have frequently observed the rhythmic contractions of the esophagus about the cardia, but I have always gained the impression that they were coincident with the respiratory movements of the diaphragm. This rhythmic contraction of the cardia, which I have at times tried to record on the kymographion, is not constant; in fact, it was not observed in the large majority of cases that I have examined.

There is only one other point to which I wish to refer, and that is, to ask Dr. Cannon's views on the subject of crossed innervation, or the antagonistic action of the vagus and sympathetic on the circular and longitudinal muscular fibres of the gastro intestinal canal—I refer to the experiments of von Basch and Ehrmann on this subject.

DR. H. W. BETTMANN, of Cincinnati: I simply rise to say a few words on the subject of the possibility of an ileo-colic sphincter, to which Dr. Cannon referred. I have often demonstrated post-mortem that pumping fluid or air into the large intestine will not cause it to pass into the small intestine. This seems to prove that the ileo-cæcal valve acts automatically, instead of possesing vital muscular action. It requires a good deal of pressure, in fact, it is necessary actually to rupture some of the muscle fibres of the inner tenia before fluid or air can be forced from the large intestine into the small. Most of these experiments were made many hours after death. We should not infer the presence of an actual sphincter at this point unless its fibres can be clearly demonstrated.

DR. L. A. BENEDICT, of Buffalo: In reference to the anti-peristaltic action of the intestines I would like to mention a case that I saw in 1888. I was asked by the late Dr. Henry F. Formad, of Philadelphia, to make a post-mortem examination in the case of a woman, rather advanced in years, who, for a week or two before her death had repeated attacks of vomiting, the material vomited having a hæmorrhagic and at times a gelatinous appearance. On section, we found the stomach and intestine partially filled with this material. Upon investigating lower down, we came upon the explanation. A large and characteristic ovarian cyst had become hæmorrhagic and was filled with the material that had been found in the patient's stomach and intestines. The cyst had become adherent to and had perforated into the transverse colon, and in this way the cystic contents found an entrance into the intestines and stomach, and had had been vomited for a couple of weeks before the patient's death.

Dr. Cannon's observations are extremely interesting, and I believe they will have a very practical application in connection with those cases in which we find a relaxation of the cardia and evidence of spasm at the pylorus, or in which, without any explanation, we find the stomach empty say half an hour after the ingestion of a test meal, the gastric contents having apparently been swept into the intestine. In dealing with stomach troubles, we may encounter so many curious combinations that the matter of therapeutics is often very puzzling, and every strictly scientific paper, like that presented by Dr. Cannon, may help us in the practical solution of the problems we have to deal with.

DR. CANNON: Dr. Hemmeter spoke of crossed innervation. That point has been worked over with great thoroughness by Langly and Anderson, and by Bayliss and Starling, but they were unable to demonstrate such an arrangement of the innervation. There are investigators, *however,* who accept crossed innervation as a settled fact.

In reply to what Br. Bettmann said, I wish to state that when we study the ileo-cæcal valve in the dead body, we have to deal with the conditions that differ materially from those in the living. Filling the colon under considerable pressure, as was done in the experiments to which Dr. Bettmann referred, is probably not the usual way in which that section of the intestinal canal is acted upon in the ordinary movements in the living subject; and furthermore, although we find it difficult to present positive morphological evidence of the existence of an ileo-colic sphincter, even in dogs or cats after death, yet in the living animal we find indisputable evidence of a sphincter at that point, but the evidence is physiological evidence. It does not seem to me that the demonstration of a mechanical valve at the ileo-colic junction in the dead human body is any proof that there is not a functional sphincter there as well. On the other hand, I have no proof that there is one; the question is still open.

I quite agree with Dr. Einhorn that the action of the pylorus may be different in pathological conditions of the stomach. But the normal influences governing the action of the pylorus are still vague; we must solve the problem from a physiological standpoint before we can attack it intelligently under pathological conditions.

THE PRESIDENT, DR. S. J. MELTZER: For my own protection, I would like to call attention to the difference between the term "crossed innervation" used by von Basch and his pupils and the "law of contrary innervation" formulated by me. Some of the facts brought out by Dr. Cannon might be explained on the basis of the law of contrary innervation, which says that the peristalsis at any point in the gastro-intestinal canal consists of two factors, namely, a contraction of the upper part, and inhibition of the lower part. For the food to progress downward, it is necessary that the path below should be opened, while at the same time it is forced down from above. The simultaneous contraction above and relaxation below, can be produced at any point of the alimentary canal by mechanical, chemical or other stimuli. Acid, for example, acts as such a stimulus. While in the stomach, it causes a contraction of the pyloric part of the stomach, and a relaxation of the pylorus itself. When it leaves the stomach it causes a contraction of the pylorus and relaxation of the duodenum. The mechanism which Dr. Cannon has observed might thus be only a part of what I have described as contrary innervation, and is, of course, I repeat, entirely different from crossed innervation.

I think that the experiments made on dead bodies alluded to by Dr. Bettmann to disprove the presence of an ileo-colic sphincter at the ileo-colic junction are not entirely conclusive. Anti-peristalsis of the right part of the colon, observed by Dr. Cannon, not only requires contraction of the colon, but also relaxation of the muscular fibres of the sphincter which may occur during life, whereas after death the resistance of the sphincter could not be overcome on account of the presence of rigor mortis in its *muscle fibres*.

[Reprinted from THE MEDICAL NEWS, May 20, 1905.]

RECENT ADVANCES IN OUR KNOWLEDGE OF THE CHEMICAL PROCESSES OF DIGESTION.[1]

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THE development of the modern physiology of digestion is almost coincident with the lifetime of our immediate predecessors. It is true that the doctrines of the older writers, the theories of concoction, putrefaction, trituration, fermentation and maceration, early yielded to the criticism and experimental deductions of Spallanzani, who, before the close of the eighteenth century, gave a firm footing to the chemical theories of to-day. But the really modern formulation of the significance of the digestive changes in the alimentary tract dates from the classic work of Tiedemann and Gmelin[2] published in 1826.

In attempting to appreciate the status of this branch of physiology at that period it is instructive to recall the prize problem announced by the French Academy in 1823. After reference to the inexact and superficial notions prevalent at that time with regard to the phenomena involved in the digestive work of the stomach and intestine, and in recognition of the advances then made in the more accurate analysis of animal and vegetable substances, the following subject was proposed for investigation, namely: "A determination, by a series of chemical and physiological experiments, of the phenomena which occur in the digestive organs during the act of digestion." The competitors were directed to investigate first the chemical or other modifications which the proximate organic principles undergo in the digestive organs, preference being given to those substances, such as gelatin, albumin, sugar, etc.,

1 Read before the American Gastro-enterological Association, New York, April 24, 1905.

2 Tiedemann and Gmelin: Die Verdauung nach Versuchen, Heidelberg, 1826.

which enter into the composition of the food. Investigation was next to be directed to the aliment itself, with due regard to distinctions between liquid and solid food; and, finally, the experiments were to be extended through "the four classes of vertebrate animals."

The problem presented by this learned body is as comprehensive as the field of digestion; and it involves many questions which still await their final solution. But the mere statement serves to illustrate how rudimentary and inadequate the conception of digestion must have been in the minds of the French scientists who proposed a subject which was well adapted to tax the ability and resources of a host of later investigators.

For several reasons it seems to me not inappropriate to refer to these facts of purely historical interest on this occasion. In an era of great progress and splendid achievements in many branches of medicine, and during a period when new aspects of science are created and developed within a single decade, when the world marvels at the revelations of modern bacteriology, and the possibilities of preventive medicine have revolutionized the modes of living, the members of this Association are liable to forget the enormous strides which that branch of physiology in which their own interests so largely center has made during three-quarters of a century. In the rapid bounds with which the study of digestion is advancing lies the justification for an attempt to present some of the more recent contributions which physiological chemistry has afforded.

From whatever standpoint the chemical processes are reviewed, the enzymes, or soluble ferments, demand the most important place among the active agents involved. The question as to their exact chemical nature is still unsolved; but with the increasing attempts to interpret physiological reactions in the light of the modern physical chemistry instead of the more obscure "vitalistic" conceptions there is a growing tendency to regard enzymes as special types of catalytic agents, colloidal in nature, which are

subject to the general laws applicable to the inorganic catalyzers. For the present, however, we are scarcely justified in going further than to direct attention to the marked correspondence between catalysis and enzyme action in respect to the laws of velocity of reaction, optimum temperature and dependence upon external influences. The so-called reversible action of enzymes deserves special notice, since it throws new light upon the synthetic processes in the organism. An enzyme solution, for example, which is able to bring about a partial hydrolysis of fats can, under appropriate conditions, rebuild these fats from their cleavage products.[3] Similar reversions have been obtained with sugar-splitting enzymes. If proteolytic enzymes are found to demonstrate a reversible action and thus to be able to resynthesize proteids from the products of proteolysis, an important addition to our knowledge of anabolism will have been made.[4] It remains to be seen whether all enzymes exhibit reversible actions, before any wide-reaching conclusions can be formulated. We may nevertheless recognize in these observations a step toward a possible explanation of the building-up or assimilative activities in the body.

The researches of recent years have taught us that enzymes cooperate in the alimentary digestive processes under conditions which have heretofore not been appreciated, and we have learned to recognize the existence of entirely new types of soluble ferments. It is a current impression that fats are not attacked in the stomach and that the gastric juice is not endowed with lipolytic properties. Although experiments reported years ago from Ludwig's laboratory indicated the possibility of the cleavage of fats in that organ, the results were generally attributed to bacterial action. Vaughan Harley and others found both free fatty acids and soaps in the stomach of the dog after ingestion of milk; and they attributed

3 Cf. Kastle and Loevenhart: American Chemical Journal, 1901, 24, 491; Hanriot: Comptes rendus, 132.

4 Cf. Herzog: Zeitschrift für Physiologische Chemie, 1903, 39, 305.

to the stomach the power to liberate fatty acids from neutral fats, thus explaining in part the relatively slight effect of the removal of the pancreas, with the attendant loss of its fat-splitting enzyme, on the digestion of fats under such experimental conditions. But it is owing to the observations of Volhard,[4] in Riegel's clinic, on human gastric juice that interest has again been aroused with respect to the gastric digestion of fats. He has shown that in emulsions of fat prepared from yolk of egg, cream and various oils, fatty acids are readily liberated by glycerin extracts of the mucosa as well as by the expressed gastric juice. The enzyme itself is very sensitive toward pepsin, hydrochloric acid and heat, and its effective action seems to be conditioned largely by the perfection of the emulsion of the fat. Moreover there is, further, some evidence of the existence of a zymogen of the stomach lipase (or steapsin).

In this connection mention may be made of a peculiar observation which has very lately been reported from Pawlow's laboratory in St. Petersburg by Dr. Boldireff.[5] In dogs with gastric fistulæ he has noted a regurgitation of a mixture of bile, intestinal and pancreatic juices into the stomach under the following conditions, namely: during prolonged hunger; after feeding with various types of fat foods; and with excess of acids in the stomach. Since it is known that fatty foods inhibit markedly the motor and secretory functions of the stomach, this investigator concludes that the digestion of such ingesta in the stomach may be dependent largely upon pancreatic juice which enters in the manner recited. If these observations receive experimental verifications in man, our conceptions regarding the chemical factors in gastric digestion may require modification. In consideration of the great number of examinations of the gastric contents constantly made by clinical observers in all parts

4 The literature is reviewed by Stade: Hofmeister's Beiträge zur chemischen Physiologie, 1903, 3, 292; see also Kastle and Loevenhart: American Chemical Journal, 1901, 24, 491.

5 Boldireff: Zentralblatt für Physiologie, 1904, 18, 475.

of the world it seems unlikely, however, that the occurrence in the human stomach of conditions comparable with those described by Boldireff would have escaped notice.

A further contribution to our knowledge of the digestion of fats is found in Boldireff's[6] discovery of an active fat-splitting enzyme in the intestinal secretion of dogs. If this shall be found to be of universal occurrence in the higher animals, it will, in conjunction with the newly noted observations on fat cleavage in the stomach, serve to explain the relatively good utilization of the fats of milk observed under conditions in which the cooperative action of the pancreatic juice is excluded.

More extensive are the additions lately made to the literature of the proteolytic enzymes. Cohnheim's[7] discovery of *erepsin* deserves detailed mention. This soluble ferment furnished by the small intestine is capable of further splitting the primary cleavage products of proteids,—albumoses and peptones,—into the crystalline nitrogenous derivatives. It does not act on the native proteids (with the exception of casein); and in this respect it differs from trypsin. Though abundant in the mucosa, erepsin is present in the intestinal secretion in small quantity only; and according to Cohnheim its action is presumably exerted upon the products of proteolysis in their slow passage through the epithelial cells during absorption. The occurrence of erepsin in the intestine has repeatedly been confirmed even in man; and Vernon[8] reports that it is present in all animal tissues, and that the various tissue erepsins are to some extent specific. It is perhaps too early to fortell the significance of erepsin for the intestinal digestion of proteids. Experimental data collected by E. Zunz[9] on dogs with ligated pancreatic ducts in-

6 Boldireff: Zentralblatt für Physiologie, 1904, 18, 460.
7 Cohnheim: Zeitschrift für physiologische Chemie, 1901, 33, 451; 1902, 35, 124; 1902, 36, 13.
8 Vernon: Journal of Physiology, 32, 50.
9 Zunz: Mémoires de l'academie royale de médecine de Belgique, 1904; cf. also Weekers: Archives internationales de physiologie, 1905, 2, 49.

dicate that it may become a valuable adjunct to trypsin. The lack of pancreatic juice may be more or less completely compensated by a more vigorous intervention of the gastric enzymes on the one hand, and of erepsin on the other.

From what has been said it is evident that suitable provision is made in the alimentary canal for a more complete breaking down of the foodstuffs than was formerly assumed. An increasing acquaintance with new cleavage products of the proteids has led to the finding of these relatively simple compounds at various times among the gastro-intestinal contents. This is, at least, true for the small intestine, the large intestine apparently retaining the function of an organ of absorption as has long been assumed.[10] Beyond the intestinal wall, in the blood and lymph stream, the cleavage products seem for the most part to be missing; and we must ask ourselves whether an extensive disintegration of the complex proteid foodstuffs actually takes place during life, and if so, what is the fate of the fragments formed. The ultimate digestion products do not accumulate in the intestinal walls; and in view of recent observations of von Bergmann and Langstein[11] it is possible that the failure to discover them in the blood is due to the fact that their disposition and assimilation in the tissues keeps pace with absorption from the intestine. I have said elsewhere that there is something attractive in the theory which assumes a more complete breakdown of the foodstuffs prior to their assimilation into living tissue or circulating blood constituents. It is not difficult to conceive how the organism can construct from these fragments the tissues peculiar to itself and accordingly maintain its chemical integrity, although the ingesta may vary widely in composition—in other words, how foreign flesh- and cereal- and milk-proteids may all give rise to the same serum albumin or

10 Cf. Glaessner: Zeitschrift für klinische Medizin, 1904, 52, 361.

11 von Bergmann and Langstein; Hofmeister's Beiträge zur chemischen Physiologie, 1904, 4, 30; cf. also Folin: American *Journal of* Physiology, 1905, 13, 126.

muscle globulin. We may look for more definite knowledge on this subject in the near future; it is a fundamental problem in physiology.[12]

It is impossible in this short paper to refer to all the agents which act upon the foodstuffs. That the intestinal juice contains enzymes capable of inverting disaccharides like cane sugar and maltose has received repeated verification in man. Some inversion may even go on in the stomach.[13] The importance of the bile as a solvent for fatty acids and soaps formed in the digestion of fats is now generally admitted, and there is a growing tendency to question the possibility of fat absorption without the intervention of digestive changes. Although the occurrence of a proteolytic enzyme in the bile of man has been reported at various times,[14] the newer animal experiments of Rosenberg[15] confirm the older observations of Voit and Röhmann in denying any important significance to this secretion in the digestion and utilization of proteids.

Two newer features of the chemical physiology of enzymes deserve mention, namely, the question of the specificity of the soluble ferments and the existence of anti-enzymes. In studying the nature of the resistance which intestinal parasites offer to the digestive juice in which they are bathed, Weinland[16] has come to the conclusion that they elaborate specific substances which can inhibit the action of pepsin and trypsin,—anti-enzymes, precipitable, thermolabile, and resembling familiar antibodies in various respects. In extending these observations to a consideration of the resistance which the stomach and intestine offer to the digestive power of the juices contained within them, Weinland[18] further found that extracts of these organs, like extracts of intestinal parasites,

12 For a recent discussion of the subject see Abderhalden: Zeitschrift für physiologische Chemie, 1905, 44, 17; Folin: American Journal of Physiology, 1905, 13, 126.

13 Cf. Ferris and Lusk: American Journal of Physiology, 1898, 1, 277; Lusk: ibidem, 1903, 10, xxi; Widdicombe: Journal of Physiology, 1902, 28, 175.

14 Cf. Tschermak: Zentralblatt für Physiologie, 1902, 16, 330.

15 Rosenberg: Engelmann's Archiv für Physiologie, 1901, 528.

16 Weinland: Zeitschrift für Biologie, 1903, 44, 1.

18 Weinland: ibidem, page 45.

check the proteolytic power of pepsin and trypsin in artificial digestion trials. We are thus brought to the recognition of a new factor in the immunity of the body against its own secretions, and its possible bearings on the pathology of the alimentary canal can only be surmised at present. To what extent, for example, is the development of certain types of gastric ulcer (Ulcer rot. ventric.) associated with absence or insufficient content of antipepsin in the gastric cells?

The foregoing observations on anti-enzymes have led to a widespread interest in their phenomena. New illustrations continue to be reported from time to time, and strengthen the probability that we are dealing here with a protective response of peculiar significance. Thus the antitryptic action of blood serum has been pointed out by various investigators,[19] and in conformity with the experience gained in the modern study of toxins, specific anti-enzymes have been developed in animals—an anti-amylase for the blood amylase,[20] antirennin, anti-antirennin[21] and antikinase[22] serving as illustrations. The suggestions involved are too new and incomplete to receive a hasty acceptance; they serve, however, to illustrate a recent tendency in the experimental study of the problems of digestion. Still more inconclusive are the recent attempts to demonstrate a far reaching specificity in the enzymes with which the foodstuffs are brought into contact. Vernon[23] and Pollak[24] have argued for a series of trypsins and erepsins; Ascoli and Bonfanti[25] postulate the existence of a multiplicity of amylolytic enzymes exhibiting noticeable preferences for specific starches. It is evident that these investi-

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19 For the literature see Oppenheimer and Aron: Hofmeister's Beiträge zur chemischen Physiologie, 1903, 4, 279.

20 Cf. Ascoli and Bonfanti: Zeitschrift für physiologische Chemie, 1904, 43, 156.

21 Cf. Korschun: ibidem, 1903, 36, 141.

22 Cf. Dastre and Stassano: Archives internationales de physiologie, 1904, 1, 86.

23 Vernon: Journal of Physiology, 29 et seq.

24 Pollak: Hofmeister's Beiträge zur chemischen Physiologie, 1904, 6, 95.

25 Ascoli and Bonfanti: Zeitschrift für physiologische Chemie, 1904, 43, 156.

gators have been influenced by the pluralistic theory of antibodies advocated so vigorously by Ehrlich.

Since the enzymes which have been discussed are the products of the digestive glands we may appropriately pause to consider briefly the development of our ideas of secretion. The purely mechanical conception of physiological processes was exemplified typically in the writings of Ludwig. Physical forces are called into play wherever possible, and filtration and diffusion assumed to explain the essential features of secretion. Ludwig and Claude Bernard in particular emphasized the important rôle of the nervous elements and vascular supply of the secretory glands; to Heidenhain we owe the more comprehensive consideration of an additional factor,—the living cell,—as the intermediator in a large number of physiological changes. He demonstrated the morphological changes which the glands undergo during activity and made it impossible longer to conceive of the secreting structure as simple diffusion membranes. His classic monograph on secretion marked a new era in the history of this subject. It established the significance of cellular activity in the work of glands, without, however, furnishing an adequate explanation of the forces which the living cell employs. Heidenhain wrote in 1883: "Je mehr sich unsere Kenntnis vertieft hat, desto klarer und zweifeloser ist es geworden, dass in den verschiedenen Absonderungsorganen die wirksamen Apparate ausserordentliche Verschiedenheiten darbieten, so dass wenigstens vorläufig die gemeinschaftlichen Merkmale viel zu allgemeiner Natur sind, um eine allgemeine Theorie der Absonderungsvorgänge zu ermöglichen."[26] The direction of progress in the succeeding twenty years is well indicated by Hofmeister in a recent lecture on the chemical organization of the cell. "Schon heute," he writes, "darf man sagen, dass die Betrachtung der Zelle als eine mit chemischen und physikalisch-chemi-

26 Heidenhain: Hermann's Handbuch der Physiologie, 5, 4.
27 Hofmeister: Die chemische Organization der Zelle, page 29.

schen Mitteln arbeitende Maschine nirgends zu Probleme führt welche die Annahme anderer als bekannte Kräfte unvermeidlich erscheinen liessen, und dass, soweit abzusehen, hier für jene Resignation, die sich einmal in einem 'Ignorabimus,' das andere Mal in vitalistischen Schlussfolgerungen äussert, kein Anlass vorliegt."[27]

The most important contribution of the last decade to the physiology of digestion lies in the newer conception of the mechanism and chemical regulation of secretion, and the correlation of the various organs engaged in this part of nutriton. The activity of the salivary glands is excited reflexly according to the nature of the food present in the mouth. The mucous membranes are not indifferent to the composition of the ingested substances and they seem to act as if endowed with discriminating sensibilities for the different foodstuffs. In the case of the gastric juice the evolution of our ideas of its secretion has been singularly progressive. Tiedemann and Gmelin, Beaumont, Bidder and Schmidt, Kühne and even Heidenhain believed to a greater or lesser extent in the possibility of mechanical stimulation of the gastric mucosa. Despite an occasional protest that true gastric juice cannot be obtained by mere contact of inert substances with the stomach membrane, it remained for Pawlow[28] and his coworkers to demonstrate that mechanical irritation either with a rod or feather or spray of sand will evoke the flow of nothing more than an occasional discharge of alkaline mucus. We have been taught from the experiments of the St. Petersburg school to look upon gastric secretion as dependent upon nervous control in which the most important reflex impulses arise in the mouth. According to Pawlow it is not the mechanical act of chewing the food nor even to any extent the chemical make-up of the ingesta which primarily induces the flow of gastric juice, but rather the psychical factor. "Appetit ist Saft!" When once the gastric secretion has been inaugurated by the psychic moment, other stimuli inter-

28 *Pawlow*: The work of the digestive glands.

vene to continue the flow; they are, however, conditioned by the chemical make-up of the food and not by the mere mechanical irritation which their presence in the stomach provokes.

An opportunity has lately been afforded to test in man the conclusions drawn by Pawlow from experiments on dogs. Dr. Hornborg,[29] of Helsingfors, has made a careful study of the conditions inducing secretion in a five-year-old boy in whom a gastric fistula was established because of a complete stricture of the esophagus. The events within the mouth could accordingly only influence the stomach membrane through nervous or circulatory channels. It was observed that the chewing of indifferent substances (like rubber) or of distasteful ones, failed to provoke secretion, whereas the mastication of appetizing foods was followed by a more or less active flow in the empty stomach. A purely psychic secretion, such as Pawlow provoked by exhibition of food, failed to be manifested in the boy. But Hornborg does not regard his trials as conclusive against the psychic theory. Still more recently comparable results on man have been reported by Umber[30] and Bickel.[31]

What are the excitants of pancreatic secretion? It has long been known that the introduction of acid into the duodenum provokes a flow of pancreatic juice. Pawlow supposed that this is due to a nervous reflex. But Popielski and Wertheimer and Lepage demonstrated that the same phenomenon may follow even after exclusion of nervous impulses from without the organ involved; and they in turn attributed the response to a peripheral reflex independent of the control of the central nervous system, but connected with local ganglia. To Bayliss and Starling[32] is due the discovery that we are here dealing with an

29 Hornborg: Skandinavisches Archiv für Physiologie, 1904, 15, 209.

30 Umber: Berliner klinische Wochenschrift, 1905, No. 3, 56.

31 Bickel: ibidem, No. 3, 60.

32 An admirable review of the subject is given by Bayliss and Starling in their Croonian Lecture: Proceedings of the Royal Society, 1904, 73, 310. On secretin see the writer's article in the Reference Handbook of the Medical Sciences, edited by A. H. Buck.

entirely different and hitherto unrecognized order of phenomena and that the secretion of the pancreas is normally called into play not through extrinsic nervous channels at all, but by a chemical substance which is formed in the mucous membrane of the upper parts of the small intestine under the influence of acid and is carried thence by the blood-stream to the gland cells of the pancreas. The name *secretin* has been given to the substance as not yet completely isolated and identified, which is the specific excitant of the gland. It is obtained by extracting the mucous coat of the intestine with 0.4 per cent. hydrochloric acid. A very small portion of such an extract injected into the circulation suffices to call forth a copious flow of pancreatic juice. The active agent is not the hydrochloric acid, since this alone does not provoke secretion when introduced directly into the blood current. The substance is not like the ordinary enzymes; its solutions can be boiled and neutralized without undergoing a diminution in activity. Secretin has already been obtained from many animals. It is not specific for the species from which it is prepared; but it is characteristic in origin since it has been impossible to obtain a body resembling secretin from any tissue of the body other than the mucous membrane of the duodenum or jejunum. The action of secretin is not confined to the pancreas; the production of bile is simultaneously stimulated and thus the factors essential for the proper intestinal digestion of the fats in particular are created.

The discovery of secretin marks a noteworthy step in advance in the study of digestion; for it has taught us a new method of chemical regulation of the secretory processes, supplementing or even supplanting the influence of the nervous system. Fleig[33] has lately found that secretin can be developed by contact of soaps, alcohol and other substances with the intestinal mucosa. From his researches it seems as if many of the

33 Fleig: Archives internationales de physiologie, 1904, 1, 286.

substances such as alcohol, ether, mustard, chloral, etc., which are known to excite pancreatic secretion may exert a twofold effect, acting in part through the intermediation of the secretin formed—the humoral path—and in part through nervous reflex ways.

We are now more easily enabled to form some synthetic conception of the events which succeed each other in the digestive cycle. The stomach contents with the combined hydrochloric acid and food masses poured into the duodenum through the relaxed pylorus, provoke a flow of pancreatic juice, bile and, according to recent observations of Delezenne and Frouin,[34] of enteric juice. An excessive discharge of acid chyme is prevented by the reflex pyloric mechanism which keeps the sphincter closed so long as the duodenal contents are acid. Meanwhile the discharged acid chyme is becoming neutralized by the flow of alkaline secretions which its very presence has inaugurated. The neutralization is again followed by the removal of pyloric inhibition and a further escape of acid chyme into the gut is permitted. Thus the double mechanism, partly chemical and partly nervous, maintains a correlation of the digestive activities and provides that the gastric contents shall be discharged only in proportion as they can be dealt with by the secretory mechanism of the intestine.

The pancreatic juice, as it is secreted, contains little if any proteolytic enzyme; in its place the zymogen, trypsinogen, is present. To Schepowalnikow,[35] a pupil of Pawlow, is due the distinction of having discovered a remarkable property of the intestinal juice in "activating" trypsinogen; that is, of converting into trypsin the zymogen of the pancreatic juice. This action is due to a peculiar enzyme, enterokinase; and the discovery introduces us to a new class of enzymes, the kinases or zymolysins, capable of activating

34 Delezenne and Frouin: Comptes rendus de la société de biologie, 56, 319.
35 Quoted by Pawlow: Das Experiment, etc., Wiesbaden, 1900.
36 Glaessner: Zeitschrift für physiologische Chemie, 1904, 40, 478.

zymogens. The observations have already been confirmed in man. The pancreatic juice collected by Glaessner[36] directly from a fistula contained no preformed trypsin, but could readily be "activated" by intestinal extracts; and Hamburger and Hekma[37] who had opportunity to collect human intestinal juice, found that it contained enterokinase. The amylolytic and lipolytic enzymes of the pancreas are apparently preformed in the secretions and require no activating agent. As an adjunct to tryptic digestion in the gut the intestinal juice has thus acquired added importance. The possible pathological consequences of an unchecked secretion of both pancreatic and intestinal juices into an empty intestine need only be hinted at. Experimentally, marked erosions have already been produced thereby under appropriate conditions. From another standpoint Popielski[38] has insisted that in disease involving the mucosa of the small intestine where the secretion of an active enteric juice is impossible, the exhibition of food is useless; for it is no longer digested. Under such conditions abstinence from food with simultaneous administration of intestinal sedatives must form the basis for rational treatment.

In addition to these chemical regulations of the secretory processes we have lately learned of the occurrence of remarkable functional adaptations on the part of the digestive glands to the work which they are called upon to accomplish. Maly wrote, in 1881: "The foods which we consume day by day are so varied in respect to odor, color, flavor and composition that it seems as though the processes of digestion are subject to daily change in every individual." Great diversity in the compositon of the digestive secretions of the different animal species was also recognized to exist; but no adequate explanation for these differences was forthcoming so long as the attempt was made to reduce the problem to broad generalizations. Pawlow and his pupils believed that they had discovered a difference in the digestive

37 Hamburger and Hekma: Journal de physiologie, 1904, 4.
38 Popielski: Centralblatt für Physiologie, 1903, 17, 70.

power of the pancreatic juice corresponding with the digestive demands of the diet used. Their conclusions, though widely quoted, have been called into question seriously by Popielski and Bayliss and Starling. Weinland[39] furnished the first convincing evidence that adaptation of the pancreas does occur. He found that while the pancreas of adult dogs contains no *lactase*—the enzyme capable of splitting up milk-sugar into monosaccharides—the extracts of dogs fed for several days on milk and milk-sugar, invariably contained lactase in considerable amount. When dogs were fed meat with the addition of milk-sugar the pancreas contained lactase, whereas milk-sugar injected subcutaneously for several days was without effect. These facts have been verified by Bainbridge,[40] and the mechanism of the adaptation investigated. The formation of lactase is a specific reaction to lactose and occurs only when the milk-sugar is given by way of the mouth. The enzyme is formed by the pancreas itself and represents a definite adaptation to a milk diet on the part of that gland. The subcutaneous injection of extracts of the intestinal mucous membrane of a milk-fed dog into a biscuit-fed dog causes the pancreas of the latter to produce lactase; this is the only means, apart from a milk diet, by which the pancreas can be made to manufacture lactase. Extracts of the intestinal mucosa of biscuit-fed dogs are ineffective. This adaptation of the pancreas to milk-sugar is carried out by a chemical mechanism. The sugar acts upon the intestinal mucosa to produce some substance which is carried in the blood-stream to the pancreas and stimulates that organ to form lactase. It is interesting to note in this connection that the pancreas of new-born puppies contains no lactase, although the enzyme makes its appearance in a few days after feeding begins.

With this glance at the mechanism by which a digestive gland is adapted to the nature of the

39 Weinland: Zeitschrift für Biologie, 38, 16, 607; 40, 386.
40 Bainbridge: Journal of Physiology, 1904, 31, 98.

food a host of questions are brought into view. What is the nature of the substance which is formed in the mucous membrane? Are other qualitative adaptations of enzymes carried out in similar fashion? To what extent can the glandular functions be altered by radical changes in diet? We may confidenly await a satisfactory solution of these related problems; at any rate the observations recorded have made clear "the intimate relation which must exist between the chemical activities of very different organs of the body."[41]

Perhaps the most striking feature of the present development of the study of digestion is the increasing complexity with which the explanation of this function is attended. Yet the unraveling of the details of these interrelated chemical and physiological reactions gives promise of a clearer understanding of the ensemble of processes termed digestion. We are rapidly learning to appreciate the significance and mechanism of the specific responses of the digestive glands under a great diversity of conditions. In 1833 Dr. Beaumont wrote in the preface to his observations on Alexis St. Martin: "The reader will perceive some slight seeming discrepancies, which he may find it difficult to reconcile; but he will recollect that the human machine is endowed with a vitality which modifies its movements in different states of the system, and probably produces some diversity of effects from the same causes." In proportion as these "seeming discrepancies" are found to be characteristic responses to definite conditions and are subjected to rigorous experimental analysis, the physiology of digestion is becoming a more exact science.

41 See also Fleig: Archives internationales de physiologie, 1904, 1, 341.

DISCUSSION.

DR. P. A. LEVENE, of New York City: Dr. Mendel's paper on this subject has been so clear and exhaustive that it is difficult to add anything to it. I perfectly agree with him that the progress made within the past few years in our knowledge of enzymes probably exceeds that made in any other field of biological chemistry.

Notwithstanding this, the present knowledge of proteid digestion and assimilation is very imperfect. Proteolytic enzymes are known to be present practically in all organs, but their role in the mechanism of food assimilation is not known; their relation to pepsin and trypsin is not fully established. In fact, it has become more difficult to make a sharp distinction between pepsin and trypsin, the products of proteolysis by either of the two enzymes being identical.

Still greater is our ignorance regarding the process of proteid assimilation. The path of proteid absorption is not definitely known, the degree of decomposition, which albuminous matter has to undergo before it can be transformed into cell and tissue substance, is not yet established. However, recent research furnished new methods which may be of great value for the solution of these problems. I shall refer here only to the "precipiitine" test. It has been established that when a proteid of foreign origin is introduced into the organism of an animal, subcutaneously or intravenously, a peculiar property is developed in the blood of that animal. The blood serum of that animal has acquired the power to form a precipitate with a solution of the proteid employed for injections. This peculiar precipitating power is not developed when the animal is fed on moderate quantities of the same proteid. This observation justifies the conclusion that under normal conditions proteid food material does not enter circulation unchanged. The conclusion is further substantiated by the observation that feeding on excessive quantities of foreign proteid may cause the appearance of a precipitine in the blood of the animal.

Prof. Mendel has further pointed out the importance of the discovery of anti-enzymes. It is perhaps worth noting that these substances are present, not only in the walls of the digestive tract, but also in other tissues, and therefore also in certain food stuffs. Is it not possible that the digestibility of a given food stuff depends also on the presence or absence in it of anti-proteolytic enzymes? By heat and by acid these anti-bodies are generally destroyed. This may be worth remembering in connection with dietary.

In conclusion I should like to make a few remarks regarding the "psychic" influence on gastric secretion. During the time that I was in charge of the Chemical Department of the Pathological Institute of the New Era State Hospital, extensive work aiming to ascertain the extent of that influence was undertaken. It was thought that the condition termed by psychiatrists, "manic-depressive insanity", may furnish suitable material for

tne investigation. There were over two hundred cases examined and recorded; however, no influence of the mental status on the condition of gastric secretion could be noted. As a rule the secretion was poor among all the inmates, and was better in those who spent much time out of doors.

DR. A. ROSE: I wish to refer to some observations in regard to the mechanical influence on the gastric juice which we have recently observed in connection with our work on Ward's Island. In dealing with cases in which there was relaxation of the abdomen with accompanying hyperchlorhydria, we found that by relieving the relaxation, the hyperchlorhydria, without any other treatment, became reduced. This was an illustration of the mechanical influence on the gastric secretion.

Dr. Levene spoke of mental influence in connection with the gastric condition, and of course we have all seen examples of it, but in the cases I refer to the influence was entirely mechanical. I think the subject is one of great importance, and since the introduction of plaster strapping for the relief of various forms of abdominal ptosis, many observations have been made showing the relations of abdominal relaxation to a number of pathological conditions. It is an established fact now that not only anomalous motive functions but also secretory disorders of the stomach may be corrected by simply relieving abdominal relaxation.

DR. MENDEL, (closing the discussion): I have very little to add. In speaking of mechanical irritation in my paper, I referred simply to the irritation on the mucosa within the stomach, rather than to any external effects. One last impression I would like to leave with the members, namely, that the more we investigate along special lines, the more we are impressed with the co-operation existing between the different organs. They are all interdependent, one influencing the other; and the various secretions depend on many factors. This fact the specialist is too apt to overlook.

[Reprinted from THE MEDICAL NEWS, July 22, 1905.]

THE IMMEDIATE EFFECT OF BILIARY RETENTION ON THE SECRETORY FUNCTION OF THE STOMACH.*

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THE fact that one digestive secretion exerts a marked influence upon another secretion has been known for a long time. Thus George Sticker[1] was the first to show that the saliva exerted a marked influence in the secretion of gastric juice and that this was of such a nature that an absence of salivary secretion not only resulted in an absence of amylolysis but that the proteolysis in the stomach was much retarded thereby. This discovery of Sticker has been confirmed by Biernacki[2] and by myself.[3]

More recently Pawlow,[4] in a beautiful series of experiments on dogs, has shown that the gastric secretion exerts a similar influence on the pancreatic secretion in so far that the former excites a flow of the latter secretion. Observations on the effect of the bile on the gastric secretion have been made by a number of observers. Hayem[5] was the first to show that usually in cases of hypertrophic cirrhosis of the liver a hyperchlorhydria and hypersecretion is found; he did not point out, however, that this condition was in any way connected with the jaundice. Leva[6] too found a similar condition in a few cases of hypertrophic cirrhosis of the liver. N. Kirikow[7] verified Hayem's results. In regard to the gastric chemistry in biliary retention various authors have obtained different results. V. Noorden,[8] Johnson and Behm,[9] Schiff,[10] Kirikow,[11] Malkow,[12] found hyperacidity, whereas Leva,[13] Schneyder;[14] Von Jaksch[15] found subacidity; others found no change whatever (Rie-

*Read at the Annual Meeting of the American Gastro-Enterological Association, at New York, April 24, 1905.

gel,[16] Swirelin[17]). The number of observations of these investigators was very small. In addition to this, inasmuch as these writers made as a rule one test of the gastric contents and rarely repeated the examination, they could not obtain a thorough account of the effect of the biliary retention on the secretory function of the stomach. It is a well-known fact that all forms of intoxication of the system can markedly influence the gastric secretion and change its character (Ankindinov,[18] Hanot et Boix[19]). A cholemic condition due to biliary retention is much like the uremic condition due to ligation of the ureter in which marked disturbances of gastric secretion are known to take place (Ankindinov [18]).

The first really systematic examination in this regard was made by S. Simnitzky.[20] He investigated especially cases of catarrhal jaundice, because in this disease the jaundice gradually increases and then gradually again diminishes. Beginning the examinations early and continuing them as the jaundice intensified and as it again diminished, the direct effect of the jaundice upon the gastric secretion could be observed. He investigated the gastric contents of the 12 patients; consisting of 7 cases of catarrhal jaundice, 1 case of Weil's disease, 3 cases of hypertrophic cirrhosis of liver, 1 case of jaundice due to compression of the common duct by a pancreatic cancer. In all 81 examinations were made. He utilized an Ewald breakfast of 35 grams of bread and 250 c.c. of tea.

He found that in those cases in which there was at first a complete biliary retention which gradually began to clear up, the total acidity as well as the percentage of free hydrochloric acid gradually diminished, and that with the retention of the bile the gastric acidity increased in proportion to the degree of biliary retention; the increase in acidity being due mainly to free HCl.

In order to investigate this condition more fully Simnitzky made a certain number of observations on dogs. He performed a gastrotomy according to Pawlow's method and studied the gastric secretion and then ligated the common bile duct and further investigated this secretion.

He found that after ligation the acidity of the gastric juice rose markedly, about 50 per cent.

My own observations include eleven cases of which eight were cases of catarrhal jaundice, one of hypertrophic cirrhosis of the liver and two of cholelithiasis; in all 115 examinations were made. In all of our observations we were careful not to include any cases in our list which were accompanied with fever or in which there was any question of an associated gastric catarrh. The gastric contents were examined for the total acidity by titration with one-tenth normal sodium hydrate solution, using phenolphthalein as an indicator; the percentage of free hydrochloric acid was determined in some cases by means of Mintz's method, in others by Töpfer's method. The cases of catarrhal jaundice were mild forms, running their course in five or six weeks. As Simnitzky points out, the variation in the state of the gastric secretion in cases of jaundice can be best observed in catarrhal icterus, inasmuch as the jaundice in this disease develops gradually, reaches a maxium of intensity and again gradually subsides, affording an excellent opportunity to observe the state of the gastric secretion during the varying intensity of the jaundice. In the eight cases of catarrhal jaundice studied as shown in the accompanying tables (See Cases 1, 2, 3, 4, 5, 6, 7, 8), similar results were observed.

The jaundice being slight at the beginning of the attack the increase in the total acidity as well as the free hydrochloric acid is found slightly above normal; gradually as the jaundice increases these factors increase proportionately and again decrease as the jaundice diminishes. So constant is this variation that we can with almost certainty predict from the condition of the acidity of the gastric secretion when the jaundice is about to diminish or disappear.

Case IX of hypertrophic cirrhosis of the liver is very interesting as showing the effect of biliary retention upon the gastric secretion in this disease. The patient, a man of forty-one years of age, had been ill of this disease for four months when he presented himself for treatment. His symptoms were marked, there was the presence of pain in the region of the liver, enlarge-

ment of the liver and spleen and an absence of ascites and the jaundice which was at times moderate and at times marked. With a moderate degree of jaundice the total acidity of his gastric contents would be as low as 70 and the percentage of free hydrochloric acid as low as .21 per cent.; while, when the jaundice was marked the total acidity would run as high as 100 and the percentage of free hydrochloric acid as high as .28 per cent.

Of even greater interest in this regard are the two cases of cholelithiasis; both of these cases were common duct cases. They were never accompanied with fever, and jaundice was always present during the attacks which would disappear again in from eight to nineteen days. In both of these cases coincident with the appearance of the jaundice there was an increase in the total acidity as high as 110 and of the percentage of free hydrochloric acid as high as .26 per cent., whereas when the attack of colic had subsided the total acidity would fall as low as 40 and the percentage of free hydrochloric acid as low as .11 per cent.

From our own observations as well as those of Simnitzky we feel justified in concluding that in conditions of biliary retention there is a marked tendency to an increase in the secretion of the hydrochloric acid of the gastric secretion. It is probable, as Simnitzky points out, that this alteration is due to a disease or asthenic state of the secretory cells of the stomach due to poisoning with biliary products.

Case I.—M. F., aged forty-five years.—CATARRHAL JAUNDICE.

No.	Date.	Total acidity.	Per cent. of free acid.	Degree of Jaundice.
1	Jan. 8	70	0.2322	Slight
2	Jan. 12	94	0.3114	Marked
3	Jan. 20	88	0.2841	Marked
4	Jan. 22	94	0.2917	Marked
5	Feb. 2	84	0.2874	Marked
6	Feb. 8	86	0.2716	Marked
7	Feb. 21	72	0.2523	Slight
8	Feb. 28	68	0.2403	Slight
9	March 22	60	0.2013	None
10	March 28	54	0.1421	None
11	April 4	48	0.1222	None
12	April 10	52	0.1316	None

Case II.—A. T., female, aged thirty-four years.—CATARRHAL JAUNDICE.

No.	Date.	Total acidity.	Per cent. of free acid.	Degree of Jaundice.
1	Dec. 5	68	0.1913	Slight
2	Dec. 15	104	0.3304	Marked
3	Dec. 23	106	0.3119	Marked
4	Jan. 14	84	0.2751	Marked
5	Jan. 25	72	0.2416	Slight
6	Feb. 10	40	0.1241	None
7	Feb. 17	44	0.1214	None
8	Feb. 28	46	0.1354	None

Case III.—F. G., female, aged twenty-two years.—CATARRHAL JAUNDICE.

No.	Date.	Total acidity.	Per cent. of free acid.	Degree of Jaundice.
1	June 4	70	0.1781	Slight
2	June 9	84	0.2659	Marked
3	July 20	70	0.2384	Marked
4	Aug. 10	42	0.1254	None
5	Aug. 18	44	0.1189	None
6	Aug. 28	40	0.1176	None

Case IV.—Male, aged thirty-one years.—CATARRHAL JAUNDICE.

No.	Date.	Total acidity.	Per cent. of free acid.	Degree of Jaundice.
1	April 4	74	0.2214	Slight
2	April 10	82	0.2703	Marked
3	April 18	88	0.2792	Marked
4	May 9	80	0.2699	Marked
5	May 19	74	0.2419	Marked
6	June 6	60	0.2120	Slight
7	June 20	44	0.1201	None
8	June 25	40	0.1324	None
9	June 28	46	0.1192	None
10	July 1	50	0.1241	None

Case V.—Female, aged twenty-four years.—CATARRHAL JAUNDICE.

No.	Date.	Total acidity.	Per cent. of free acid.	Degree of Jaundice.
1	Feb. 4	68	0.1817	Slight
2	Feb. 8	90	0.2754	Marked
3	Feb. 12	100	0.2951	Marked
4	Feb. 20	88	0.2634	Marked
5	Feb. 25	84	0.2712	Marked
6	March 12	78	0.2316	Slight
7	March 20	70	0.2219	Slight
8	April 9	50	0.1512	None
9	April 12	48	0.1229	None
10	April 18	45	0.1182	None
11	April 22	48	0.1174	None
12	April 28	50	0.1093	None

Case VI.—Male, aged forty-one years.—CATARRHAL JAUNDICE.

No.	Date.	Total acidity.	Per cent. of free acid.	Degree of Jaundice.
1	Oct. 3	74	0.1612	Slight
2	Oct. 15	86	0.2609	Marked
3	Oct. 25	80	0.2671	Marked
4	Nov. 18	88	0.2789	Marked
5	Dec. 1	70	0.2219	Slight
6	Dec. 20	54	0.1348	None
7	Dec. 28	42	0.1218	None
8	Dec. 30	40	0.1114	None

Case VII.—Female, aged forty-eight years.—CATARRHAL JAUNDICS.

No.	Date.	Total acidity.	Per cent. of free acid.	Degree of Jaundice.
1	June 10	68	0.1719	Slight
2	June 18	92	0.2846	Marked
3	June 29	110	0.2982	Marked
4	July 5	90	0.2879	Marked
5	July 12	88	0.2756	Marked
6	July 20	92	0.2874	Marked
7	July 28	84	0.2769	Marked
8	Aug. 5	88	0.2642	Marked
9	Aug. 9	86	0.2718	Marked
10	Aug. 12	78	0.2469	Marked
11	Aug. 20	70	0.2191	Slight
12	Aug. 27	60	0.1918	None
13	Sept. 3	54	0.1216	None
14	Sept. 9	52	0.1189	None
15	Sept. 13	56	0.1072	None

Case VIII.—Male, aged forty years.—CATARRHAL JAUNDICE.

No.	Date.	Total acidity.	Per cent. of free acid.	Degree of Jaundice.
1	April 4	70	0.2191	Marked
2	April 12	84	0.2684	Marked
3	April 18	92	0.2784	Marked
4	May 5	98	0.2796	Marked
5	May 9	86	0.2687	Marked
6	May 15	84	0.2695	Marked
7	June 4	78	0.2421	Marked
8	June 30	58	0.1189	None
9	Aug. 5	52	0.1213	None
10	Aug. 8	44	0.1184	None

Case IX.—Male, aged forty-one years.—HYPERTROPHIC CIRRHOSIS OF THE LIVER.

No.	Date.	Total acidity.	Per cent. of free acid.	Degree of Jaundice.
1	June 4	70	0.2219	Moderate
2	July 9	78	0.2318	Moderate
3	Aug. 5	94	0.2659	Marked
4	Aug 20	100	0.2864	Marked
5	Sept. 8	72	0.2214	Moderate
6	Sept. 15	70	0.2187	Moderate
7	Sept. 23	74	0.2089	Moderate
8	Oct. 20	80	0.2501	Marked
9	Nov. 10	84	0.2582	Marked
10	Dec. 12	82	0.2517	Marked

Case X.—Female, aged thirty-five years.—CHOLELITHIASIS.

No.	Date.	Total acidity.	Per cent. of free acid.	Degree of Jaundice.
1	Feb. 4	45	0.1182	None
2	Feb. 20	40	0.1143	None
3	March 25	80	0.2483	Marked
4	March 28	84	0.2591	Marked
5	April 2	92	0.2683	Marked
6	June 6	50	0.1382	None
7	June 12	54	0.1243	None
8	July 2	45	0.1134	None
9	Sept. 8	90	0.2684	Marked
10	Sept. 11	84	0.2517	Marked
11	Sept. 18	76	0.2481	Slight
12	Nov. 7	42	0.1243	None
13	Nov. 14	44	0.1189	None
14	Nov. 22	46	0.1146	None

Case XI.—Male, aged forty-two years.—CHOLELITHIASIS.

No.	Date.	Total acidity.	Per cent. of free acid.	Degree of Jaundice.
1	June 6	88	0.2482	Marked
2	June 12	110	0.2694	Marked
3	June 18	84	0.2514	Marked
4	Aug. 10	42	0.1345	None
5	Aug. 14	48	0.1394	None
6	Sept. 28	92	0.2487	Marked
7	Oct. 4	96	0.2416	Marked
8	Oct. 16	72	0.2386	Slight
9	Dec. 6	46	0.1387	None
10	Dec. 20	40	0.1313	None

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DISCUSSION.

DR. A. L. BENEDICT: I do not like to discuss this paper in Dr. Friedenwald's absence, but I have had quite a number of cases under my observation in which the symptoms suggested hyperchlorhydria, and which subsequently proved to be cases of biliary obstruction. Of course, there was no jaundice at the start or the diagnosis would have been easy. In those cases—I should say ten or twelve altogether—there was no free hydrochloric acid, but usually considerable organic acid and evidences of ischochymia. This brings up the question as to whether the condition of the stomach is due to the chemical effect of the retained bile, or merely a reflex from the spasm of the biliary passages. Perhaps some of the members can throw some light on the subject. My own experience includes no cases of demonstrated hyperchlorhydria in biliary retention, and the cases in which hyperchlorhydria was suspected but not found were not jaundiced.

DR. MAX. EINHORN. I must say that I have certainly seen cases of jaundice and hypertrophic cirrhosis of the liver in which there was no evidence of an increase of hydrochloric acid, but rather a diminution. In the majority of cases, the observation contained in Dr. Friedenwald's paper may be correct, but it cannot be taken as a general rule.

Sonderabdruck aus der Berliner klin. Wochenschr., 1905, No. 44a.

Are the proteolytic and milk coagulating effects of gastric and pancreatic juice due to one and the same enzyme?

By

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The distinguished St. Petersburg physiologist, J. P. Pawlow, associated with S. W. Parastschuk, has published a series of experimental investigations under above heading in Hoppe-Seyler's Zeitschr. f. physiol. Chemie, Bd. 4, p. 315 to 452. (The exact title of the article is „Ueber die ein und demselben Eiweissfermente zukommende proteolytische und milchkoagulierende Wirkung verschiedener Verdauungssäfte.") The view hitherto held and still held by most physiologists is that the proteolytic and milk coagulating effects of gastric juice (of pancreatic juice also), are due to specific enzymes. The earlier investigations on the subject date from Hammarsten (Maly's Jahresberichte, 1872, Bd. II, S. 118; see also Hammarsten's Lehrbuch der physiol. Chemie, 5th edition). In order to precisely state the direction of the inquiry it is necessary to emphasize that Pawlow and Parastschuk attempt to prove that a separate and distinct milk curdling enzyme does not exist, but that milk coagulation is only a property of the pepsin. The „Fragestellung" here is not as precise as might be desirable, for proteolytic and milk coagulating effects might be conceived as attributable to one and the same proteid ferment, and yet pepsin and chymozin may be two different enzymatic effects, for we may conceive that pepsin, the proteolytic enzyme, possesses also to a certain extent a milk coagulating effect, which is only developed in an acid reaction and not in a neutral reaction, and as far as human

gastric juices are concerned, it can be stated that such an enzyme occurs which will curdle milk in the presence of an amount of HCl which, as a rule, is normal to human gastric juice (2 parts per thousand), but will not curdle milk if the gastric juice is exactly neutralized. This disappearance of milk coagulating power in the case of canine gastric juice, after it has been neutralized with sodium bicarbonate, was noticed by Pawlow and Parastschuk (l. c., p. 422), but they attributed this disappearance to a destructive effect of the alkalies employed, particularly to Na_2CO_3. When the juice was neutralized with $NaHCO_3$, the ferment could be preserved. To the destruction of the pepsin by alkalies they attribute their inability to separate these two enzymes by Hammarsten's method (shaking of gastric juice with magnesium carbonate, by which the pepsin is torn down by this insoluble powder, whilst the chymozin [Lab] remains in solution.) The third, fourth and fifth filtrates prepared in this way will coagulate milk energetically, but will not digest raw fibrin in 24 hours not even if raised to the normal acidity. But Pawlow asserts that when this filtrate was diluted 5 to 10 times its volume with XX 0,1 to 0,2 per cent solution of HCl, the proteolytic effect on fibrin became distinctly evident.

The two enzymes, however, can be separated in a much simpler manner — by heating gastric juice to 65° C. for ten minutes, the chymozin can be destroyed. When chymozin is in solution together with HCl — 3 parts per 1000 — the enzyme can be destroyed completely if the solution be kept at a temperature of 37 to 40 degrees C. for 48 hours (Hammarsten, l. c., p. 291). The pepsin is not destroyed by this process. In this manner a pepsin solution free from chymosin can be obtained. A solution of parachymosin, free from pepsin, can also be prepared in a similar manner (Ivar Bang, Ueber Parachymozin, ein neues Labferment, Pflüger's Archiv, Bd. 79, S. 425). Bang's investigations have made it very highly probable that the chymosin enzyme of different animals are by no means identical, but show striking differences in milk coagulating power. Accordingly it becomes necessary in testing the results of previous investigators, to use the chymosin from the same animals as they employed.

In „Beiträge zur chemischen Physiologie und Pathologie", *Bd. VI, H. 8, S. 396,* P. Schrumph described a new method for *preparing pepsin* from the expressed juice of mucosa of pigs *stomachs.* Schrumph prepares four different solutions according

to the different chemical treatment to which he subjects this pressed juice of gastric mucosa. Solution **A**: minced gastric mucosa is thoroughly. cleansed of its mucosa with water, then rubbed up intimately with powdered silicon, then this mass is expressed by a Buchner press, with which gradually a pressure of 600 atmospheres is reached. From five pig gastric mucosas he thus gained 100 c. c. of a slightly opaque press juice. This is solution **A**. After this has been passed trough a Chamberland filter it becomes solution **B**., and when this again is dialyzed for 24 hours in flowing water he obtains solution **C**. Solution **C**. is perfectly clear, neutral or very slightly acid, biuret and Millon's reaction positive. Addition of alcohol causes no precipitate, but on addition of ammonium sulphate, acetic acid, picric acid, uranyl acetate, HCl, a cloudiness results. Pepsin digestion is very distinct, and even evident without the addition of HCl. The milk coagulating effect is very energetic. Now comes the important part of Schrumph's preparation. A small amount of cholesterin is added to 10 c. c. of a mixture of ether and absolute alcohol and poured in the remainder of solution **C**. A very dense floculent precipitate is produced, which is rapidly centrifuged off, thereafter filtered off, and suspended in the original quantity of water. It must then be yet shaken with smaller quantities of ether, which does not make the solution absolutely clear, but it can be made so by passing it trough a Kitasato (candleshaped, Kerzenfilter) filter. Eventually one obtains a perfectly clear filtrate. This is solution **D**. In this solution biuret and Millon's reaction are negative. Acetic acid, picric acid, HCl, uranyl acetate, ammonium sulphate, cause no cloudiness. These reactions, however, can be very distinctly produced in the fitrate obtained after the first removal of the cholesterin precipitate. This solution D. has a powerful proteolytic effect on addition of 0,2 per cent. HCl. The milk coagulating effect is absent entirely.

The comparative proteolytic and coagulating effects of Schrumph's solutions are the following:

5 c. c.	solution	A	digest in	2	hours	3 mm.	Mett;	milkcoagul.	positive	
5 „	„	B	„	„ 2	„	3 „	„	„	„	
5 „	„	C	„	„ 2	„	4 „	„	„	„	
5 „	„	D	„	„ 2	„	8 „	„	„	negative.	

The supposition that the milk coagulating effect was pre-*vented or obscured* in this case by some foreign addition, can

hardly be substantiated in view of the process of preparation. It is probable therefore that Schrumph has prepared a pepsin solution which is free from proteid, has an energetic proteolytic effect, but no milk coagulating effect whatever. If pepsin and chymosin belong to the same molecule, it is to be expected that these two effects stand in a definite and constant relation to each other with regard to their intensity of action. This Pawlow and Parastschuk endeavor to demonstrate by showing up parallelism of effects and proportionality of work done between pepsin and chymosin. Whilst parallelism and proportionality can be considered attributes of an enzyme in which proteolytic and milk curdling effects are due to the same molecule, it does not exactly disprove that the two effects may be due to two different molecules, for we can arrange solutions of two well-know enzymes with characteristic different effects, and still the work done can be demonstrated to have been parallel and proportionate. I base my view that pepsin and chymosin are two different and spezific enzymes, or in other words, that the proteolytic and milk curdling effects are due to different molecules, mainly upon the following experiments and observations: (1) Parallelism of effects and proportionality of work done by standardized solutions of two different enzymes artificially produced, or by two different digestive secretions; (2) Parallelism and proportionality as above defined observable in the effects of two different inorganic catalyzers, such as colloidal, platinum, and colloidal silver or gold; (3) The presence of only one of these effects, the proteolytic, and the absence of the milk curdling effect, in certain conditions of the human gastric mucosa; (4) The experiments of Ivar Bang and Schrumph, already described.

In experiments with milk coagulation which extend over 10 hours it must not be overlooked that milk may coagulate spontaneously even when drawn and preserved under absolutely sterile conditions. Pawlow and Parastschuk, in attempting to demonstrate that proteolytic and milk coagulating effects are attributable to one and the same enzyme, offer as proof of their assertion the parallelism of effects and proportionality of work done — the degree of proteolysis was measured in millimeters *of a column of boiled* albumen (Mett's method, devised in *Pawlow's laboratory)*, and the degree of coagulating power was *measured by the time* that was required to coagulate 10 c. c. of

milk by varying amounts of gastric juice. The coagulation of the milk always occured in an acid reaction. The acidity of the various portions of the gastric juice was equalized by 0,5 solution of HCl. In table I (l. c., p. 417), Pawlow demonstrates that the least chymosin was secreted when the test food was milk (10 c. c. of milk coagulated in 50 minutes), and most chymosin was secreted when the test food was bread (10 c. c. of milk coagulated in 2.5 minutes by the bread juice of the same animal). This same juice digested 5.8 millimeters of albumen column (Mett), showing that the proteolytic power was very active also in the same bread juice.

But on page 418, Table II, we are told that a bread juice which digested 5.8 millimeters, did not coagulate 10 c. c. of milk until 3 minutes and ten seconds had elapsed (65 seconds longer than the previous juice which had digested the same length of albumen column). In table II the coagulation time is approximately the same for bread, meat, and milk juice, 190 to 195 seconds, but the relative concentration of the ferments is placed at 1 for bread juice, 4,29 for meat juice, and 8,41 for milk juice. On page 419, the authors assert that this table II shows a perfect proportionality of proteolytic effects and of milk curdling power. The further efforts of these investigators were directed towards showing a parallelism of these two effects of gastric juice under the influence of (a) putrefaction of the juice, (b) the influence of heat, table V, and (c) the influence of chemicals, salts, bile, alcohol etc. Similar experiments are then narrated concerning pancreatic juice obtained in various manners. It is interesting to learn that the pancreatic juice as secreted was inactive for curdling milk, and had to be activated by the addition of succus entericus, and that the activation of the milk curdling agent occurs at the same rate as the activation of the proteolytic agent, under the influence of succus entericus.

On page 432 we are told that table XII, which shows the proteolytic power and milk coagulating power of juice (1) from gastric pepsin glands, (2) from pylorus glands, and (3) from Brunner's glands – gives the evidence that the two powers correspond perfectly. Table XII, however, does not show this convincingly. In column 2 of that table the proteolytic power is shown to sink from 2,5 mm Mett for pepsin gland juice, to 0,5 *mm Mett for Brunner's* gland juice. But the milk coagulating time for pepsin gland juice is 27 minutes, for Brunner's

gland juice it is 18 minutes. In order to show an exact proportionality, the milk ought to have been coagulated in the same periods of time, as is shown in table II, p. 418. But it is apparent from this table XII that as the proteolytic power sank, the coagulating power became intensified. A possibly involuntary distinction is made between these two effects on page 439, where dilution is said to favor the proteolytic power, restoring it when it had been destroyed, by the sodium acetate (criticism of Glässner's experiments in Beitr. z. chem. Physiol. und Pathol., Bd. I, 1901). But on the other hand dilution is said to destroy the coagulative power. A reduced proteolytic power und the checking influence of salts, or dilution, does not necessarily indibate that the ferment is present in smaller quantities. It may be that the substance which is to be acted upon proteolytically, the albumen of the fibrin, is so altered by the salts or other substances employed as to render them more insoluble. This can be demonstrated by performing experiments with known strengths of pepsin, allowing it to act first upon ordinary pure fibrin, and then upon fibrin which has been allowed to become saturated in a 20 per cent solution of sodium acetate and thereafter washed in distilled water. The proteolysis will be markedly less in the last case.

In the most recent edition of Lehrbuch der physiolog. Chemie, Hammarsten objects to the main conclusion of Pawlow and Parastschuk that the two effects spoken of are due to one and the same enzyme, on the ground that two different rules exist according two which these two effects depend upon the quantity of ferments. According to Pawlow, however, the time of these reactions varies with the conditions influencing them. Some may accelerate and others may prolong the time of reaction. The chief conditions which he cites are alcalinity, acidity of the medium, and the degree of dilution (concentration). The authors recognize three rules under such conditions. A neutral solution of chymosin causes milk coagulation provided it is present in the solution in a certain definite concentration; (1) then coagulation occurs according to the rule of inverse proportionality between the quantity of the ferment and the coagulation time; (2) when the chymosin is in acid solution the quantities of the ferment are inversely proportional tho the square root of coagulation time, that is, the reaction is progressively accelerated; (3) in a *very much diluted* neutral solution of ferments the quantities of

the enzymes are porportional to the squares of the coagulating time. These three varying conditions concern mainly the milk coagulating power. In a similar way Pawlow shows that the rules of the dependence of the effects of enzymes upon the amounts of the exzymes can be influenced by external conditions. All such rules are reliable only within very narrow limits.

After reading the article as very briefly abstracted in the preceding, we are justified in asking whether parallelism of effects and proportionality of work done in two different catalytic actions, really constitute satisfactory proof that the two actions are due to one and the same catalyzer. Pawlow and Parastschuk demonstrate that the proteolytic and milk curdling effects of pancreatic juice show a parallelism and proportionality under varying conditions. But the same can be shown to be true of the amylolytic and fat-splitting power of pancreatic juice. Why then not assert that the amylolytic and lipolytic effects are due to one and the same enzyme also? In the American Journal of Physiology, Vol. X, p. 191, Hugh Neilson has shown that colloidal platinum, like lipase, can bring about hydrolysis and synthesis of fats. He also shows that this hydrolysis by platinum increases with the concentration of the platinum, it increases with the temperature, it is independent of the concentration of the ethyl butyrate, and that poisons (see table, l. c., p. 197), reduce the hydrolysis by platinum black. This same variation of effects, under the influence of concentration, temperature, poisons and salts, can be shown to exist with regard tho the effects of a number of organic enzymes. For example, in case of saliva it can be shown that the amylolytic action, the conversion of starch into dextrose, is increased by concentration of the ferment, it is increased by heat, it is injured by certain chemicals, almost in the same manner as lipase is injured in its hydrolysis of ethyl butyrate (experiments of Kastle and Loevenhart, American chemical journal). If, therefore, we should execute a series of experiments in two series, ohne with colloidal platinum on ethyl butyrate under these varying conditions, and the other with saliva and boiled starch under varying conditions, certainly an exactly similar rising or falling of the amount of catalytic work done, that is, a parallelism of effects, could not be asserted as being due to one and the same catalyzer in this case, for we know that two different agants are used in the experiments. The figures

of the hydrolytic effect of colloidal platinum con giver in the article by Hugh Neilson (l. c.).

When chymosin or rennet is separated from the stomach of a calf by Blumenthal's method (see J. Reynolds Green, „The Soluble Ferments", Cambridge Natural Science Manual, 1899, p. 237), an amorphous white gelatinous substance is gained, which greatly resembles aluminum hydrate in appearance. This precipitate in nearly pure rennet, and a very small portion of it speedily causes coagulation of milk. It has no odor or taste when prepared by this method, it readily dissolves in water, forming a clear solution. Blumenthal asserts that the same treatment will separate two enzymes from the so-called pepsin essences of commerce, and that the mother liquid from which the rennet is removed has no curdling action. The trypsin and chymosin of the pancreatic juice were separated by Roberts, a salt solution extract of a pancreas was slightly acidulated with HCl and kept at 40° C. for three hours. When neutralized it was found that the trypsin had been destroyed, while the rennet or chymosin was unharmed.

W. D. Halliburton and Brodie draw a distinction between gastric and pancreatic rennet which has not been noted by previous observers. According to them the clotting indicated by pancreatic rennet is not a true coagulation, but a precipitation which takes place in a warm bath a 35°—40° C. The precipitate is finely granular, and cannot be detected by the naked eye. On cooling it to the temperature of the air, it sets into a coherent curd which can be again broken up by warming to 35° C., when the granular condition returns, and the milk appears fluid. This may be repeated several times. Halliburton and Brodie call the proteid in this condition „pancreatic casein", and they say it can be converted into tyrein by gastric rennet.

The chymosins of different animals are not identical, and the chymosin of different organs in the same animal, as Halliburton and Brodie have shown, are not identical. The differences in the chymosins of various animals are considerable (Ivar Bang, Pflüger's Archiv, Bd. 79, p. 425). Most of the experiments with chymosin or rennet made prior to the publication of Pawlow and Parastschuk, were made with the chymosin *of the calf's stomach*, and not until a comparative quantitative *study of he various* chymosins has been made, can it be proven *that the milk coagulating* power of all chymosins are alike, or

that this power is governed by the same rule with all forms of this ferment. Dilution, for instance, affects the milk curdling power of chymosin from the pig in a different manner from the chymosin obtained of the dog's stomach. For comparing the effects of their chymosins with the results of previous investigations, it would have been necessary for Pawlow and Parastschuk to use the chymosin of the same animal. Pawlow and Parastschuk, in attempting to prove that proteolysis and milk curdling effects are due to one and the same enzyme, laid greatest stress upon the demonstration of parallellism of effects under various chemical and physical influences, and secondly upon proportionality of work done. To subject a digestive secretion that gives evidence of two strikingly different effects to varying external influences, and thereby showing that the effects increase and diminish together, or disappear together, can hardly be considered a satisfactory form of experimental logic to prove that these two different effects are due to one and the same agent. The work of these authors proves that the technique and method of experimentation may be objective and perfect, and yet the subjective deduction therefrom may be capable of different interpretations. In order to make clear my meaning I will cite the example of two well-known digestive enzymes, the actions of which occur best in a mildly alkaline medium, ptyalin of the saliva, and trypsin of the pancreatic juice. Trypsin prepared by Kühne's method (Unters. a. d. physiol. Institut der Universität Heidelberg, Bd. I, 222), and ptyalin both can be shown to be augmented and inhibited in their characteristic effects upon fibrin and boiled starch respectively in a parallel manner by temperature, by chemicals and poisons, by concentration and dilution. The amount of ptyalin and the amount of boiled starch in the one case, and the amount of trypsin and the proteid upon which it acts, either fibrin or boiled egg albumen, can be so adjusted that a parallelism of effects and proportionality of work done can be shown to exist between these two ferments. If the same logic were applied to these phenomena as Pawlow and Parastschuk apply to milk curdling and proteolysis, it would be equally possible to assert the amylolysis and proteolysis to be due to one and the same enzyme, if it were *not positively* known beforehand that two definite enzymes *were employed.* The method employed in Pawlow's laboratory

to determine the degree of amylolysis is similar to that of Mett for proteolysis. Mett tubes are employed which are filled with filtered egg albumen coagulated in a water bath, and sealed at both ends with sealing wax. For amylolysis the tubes are filled with boiled starch in a similar manner. The diastatic power is expressed by the number of millimeters of boiled starch digested in a given time. In most of my work I employed pancreatic juice obtained from the duct of Wirsung of the dog.

Experiments with dog saliva and pancreatic juice trypsin (Kühne), demonstrating effects of increase of temperature. On digestion of boiled starch and albumen respectively.

Canine saliva and trypsin solutions were subjected in different and separate portions to the various temperatures stated in column 1, and thereafter their amylolytic and proteolytic power tested.

Temperature.	Saliva. No. of m. m of boiled starch column digested by.	Pancreatic juice trypsin. No. of m. m of albumen column digested.
10° C.	4,20	5,52
12° C.	4,15	5,5
15° C.	4,25	5,9
20° C.	4,25	6
30° C.	5,1	6,5
40° C.	4,95	5,9
50° C.	3,5	4,5
52° C.	2,75	3,62
54° C.	2	3
56° C.	0,5	0,9
58° C.	0,3	0,5
60° C.	0	0
62° C.	0	0
64° C.	0	0

Both enzymes exhibit a first a slight increase of their characteristic effects followed by a gradual diminution until the enzymes are completely destroyed at 60° C., a parallelism of effects — which can also be demonstrated if both saliva and trypsin solution are poured together and a starch, as well as an albumen tubule, *placed in* the combined solution simultaneously. But here the *action of amylopsin* requires exclusion.

The following tables demonstrate the gradually reducing effect of the chymosin and pepsin, and also the changes in the free HCl and total acidity in gastric juices drawn from human patients, It will be seen from these tables that parallelism of effects and proportionality of work done are as a rule not evident. The cases „Pyle", „Ward" and „Weinberg" were cases of gastritis acida, those of „Cartridge", „Hamilton" and „Dawkins" were cases of hyperchlorhydria (nervosa), the case of „Warner" was one of dilatation of the stomach, and the case of „Spencer" was one of hyperchlorhydria.

Tests for any change of acidity values in the human gastric juice on standing at room temperature.

Ward.

Date	Free HCl	Total Acidity	Proteolysis in mm. Mett tubes	Chymosin, 10 cc. milk coagulated by 0,2 cc. gastric juice in minutes
November 16	34	48	5	3' 15"
„ 17	31	47	5	3' 15"
„ 18	34	48	5	3' 15"
„ 19	34	48	5	3' 15"
„ 21	32	48	5	3' 15"
„ 22	32	48	5	3' 15"
„ 23	34	48	5	3' 15"
„ 24	32	50	5	3' 10"
„ 25	32	50	4,5	3' 20"
„ 26	32	50	4,5	3' 20"
„ 28	34	49	5	3' 24"
„ 29	32	48	4,5	5'
„ 30	32	50	4,5	5'
December 1	31	48	4	5'
„ 2	32	48	3,5	6'
„ 3	32	49	3,5	8'

Weinberg.

Date	Free HCl	Total Acidity	Proteolysis in mm. Mett tubes	Chymosin, 10 cc. milk coagulated by 1 cc. gastric filtrate in minutes
November 17	39	62	4,5	3'
„ 18	42	64	4	3' 30"
„ 19	44	63	4,5	3'
„ 21	42	65	4	3'
„ 22	42	66	4,5	3' 30"
„ 23	41	70	4	3'

Pyle.

Date	Free HCl	Total Acidity	Proteolysis in mm. Mett tubes	Chymosin, 10 cc. milk. coagulated by 1 cc. gastric filtrate in minutes
November 22	29	64	4,5	3′ 30″
„ 24	29	64	4,5	4′
„ 25	30	66	4,5	4′
December 1	30	65	4,5	3′ 50″
„ 2	31	65	4,5	3′ 50″
„ 3	30	64	4,5	3′ 50″
„ 4	30	65	4.5	3′ 50″
„ 5	31	66	4,5	3′ 50′
„ 6	30	64	5,5	3′ 20″
„ 7	31	65	5,5	3′ 20″
„ 8	31	64	5,5	3 20″
„ 9	31	64	5,5	3′ 20″
„ 10	32	64	5	3′ 20″

Cartridge.

Date	Free HCl	Total Acidity	Proteolysis in mm. Mett tubes	Chymosin, 10 cc. milk coagulated by 0,2 cc. gastric filtrate in minutes
January 17	74	100	8	5′
„ 18	76	104	8	5′ 10″
„ 20	76	108	8	5′ 10″
„ 23	74	104	8	5′
„ 25	74	104	8	5′ 10″
„ 26	76	104	8,5	5′
„ 26	72	102	7	4′ 20″
„ 30	78	106	7,5	4′ 30″
„ 31	74	106	6	4′

Increasing cloudiness of gastric filtrate made the exact time of turning of the dimethyl amido benzol more difficult to recognize as the filtrate became older. The free HCl was then accertained in other specimens by the Hoffman method (dissociation of methyl acetate) with same results.

Increasing cloudiness of gastric filtrate made the exact time of turning of the dimethyl amido benzol more difficult to recognize as the filtrate became older.

Same gastric juice of Spencer, when left in incubator at *36° C., began to lose* its proteolytic power in 20 days. The milk

Hamilton.

Date	Free HCl	Total Acidity	Proteolysis in mm. Mett tubes	Chymosin, 10 cc. milk coagulated by 1 cc. gastric filtrate in minutes
1904 December 30	65	110	6,5	4'
1905 January 3	60	110	6	4' 10"
„ 5	64	116	6,5	4'
„ 7	66	120	6,5	4'
„ 10	70	124	7	3' 10"
„ 12	62	120	6	3' 40"
„ 14	74	118	6	3' 40"
„ 18	68	118	6,5	3' 45"
„ 20	70	120	7	3' 10"
„ 22	66	118	6,5	4'
„ 26	68	118	6,5	4'

Dawkins.

Date	Free HCl	Total Acidity	Proteolysis in mm. Mett tubes	Chymosin, 10 cc. milk coagulated by 2 cc. gastric filtrate in minutes
1905 January 25	60	90	5	1'
„ 28	62	100	4,5	1'
„ 30	62	88	5	1' 5"
February 1	62	88	4,5	1' 5"
„ 4	62	98	4,5	1' 15"
„ 6	60	96	5	1' 5"
„ 8	60	100	5	1' 5"
„ 10	62	110	5	1' 5"
„ 14	62	105	5	1' 5"
„ 18	62	100	5	1 5"

curdling power diminished more rapidly and disappeared entirely on the eighteenth day.

From Spencer a very large test meal was drawn, amounting to 760 c. c. of gastric chyme, which was kept under experimentation 56 days. In this table the figures show the proteolysis to diminish gradually from 8 mm. on January, to 4 mm. on February 28, but the milk coagulating power diminishes mor rapidly and out of all proportion to the rate in which the proteolytic power diminishes. No parallelism in the diminution of these effects is here detectable.

Warner.

Date	Free HCl	Total Acidity	Proteolysis in mm. Mett tubes	Chymosin, 10 cc. milk coagulated by 2 cc. gastric filtrate in minutes
1905				
February 1	26	66	3	5' 0"
„ 2	24	70	3	5' 0"
„ 4	24	72	3	5' 0"
„ 6	26	74	3	5' 0"
„ 8	26	72	3	5' 0"
„ 10	26	72	3	8' 0"
„ 14	28	72	3,5	8' 30"
„ 16	26	70	3,5	10' 15"
„ 20	28	70	3,5	10' 15"
„ 21	26	68	3,5	11' 0"
„ 24	28	70	3,5	12' 0"
„ 26	28	68	3,5	15' 10"

One of the most interesting facts discoverable during persistant analyses of human gastric juice is the total absence in some of them of the milk curding enzyme, whilst at the same time the peptic or proteolytic enzyme is present. This is proven by the fact that whilst the filtrate from test meals consisting of bread and water will not curdle milk, it will give the biuret reaction when it is added to disks of boiled egg albumen or fibrin thereafter pleced in the digestorium. This occurs in certain stages of chronic atrophic gastritis, when the milk curdling enzyme disappears before the peptic enzyme. I have also seen it in two cases of cancer of the stomach. I must emphasize, however, that I have studied the gastric juice of two persons who were normal to all subjective and objective examinations. I have frequently, in order to emphasize the fact that the milk curdling enzyme is absent, poured a pint of milk into the stomach of one of these persons, and withdrawing it by the stomach tube an hour and 20 minutes thereafter, in the identical condition in which it was poured down through the stomach tube. Nor did this milk coagulate after it was allowed to stand in an incubator for three hours. Nor could it be made to coagulate in less time than three hours by the addition of varying amounts of calcium carbonate, nor by the addition of 1/10 normal solution of HCl added until the normal amount which should be present in the

Spencer.

Date	Free HCl (Töpher's Method)	Total Acidity	Proteolysis in mm. Mett tubes	Chymosin, 5 cc. sterile milk coagulated by 2 cc. gastric filtrate in minutes
1905				
January 3	60	64	8	30″
„ 4	60,5	66	8	30″
„ 5	60,6	64	8,5	20″
„ 6	61	66	8,5	30″
„ 7	60	66	8	30″
„ 9	60	64	8	30″
„ 12	61	65	8	30″
„ 14	60,5	65	8	30″
„ 15	61	66	8,5	30″
„ 18	61	65	8	30″
„ 20	66	60	8	30″
„ 21	65	60	8,5	30″
„ 24	65	59,5	8	30″
„ 26	65	59,5	7,5	1′ 8″
„ 28	65	60	7,5	1′ 8″
„ 30	60	65	7,5	1′ 8″
„ 31	59	65	7,5	1′ 8″
February 1	60	65	6	1′
„ 3	59	66	5,5	1′ 2″
„ 6	59	66,5	5	1′ 2″
„ 10	58	67	5	2′
„ 14	57	68	5	3′ 5″
„ 16	55	67,5	5	3′ 5″
„ 18	50	68	4,5	4′ 0″
„ 20	50	69	4,5	5′ 5″
„ 22	48	70	4	10′
„ 24	48	72	4	12′ 5″
„ 28	46	74	4	20′ 5″

gastric juice is reached. Milk to which HCl is added in this manner will coagulate when no enzyme is present but it takes a much longer time (10—16 hours). Gastric juice which was boiled, and thus had its enzymes destroyed, did not coagulate milk any sooner than a solution of HCl of the same strength. In testing milk coagulation on the addition of HCl, the time in which the coagulation occurs is an important factor in the determination. The same persons whose gastric juice could not curdle milk produced a feeble proteolytic enzyme, for the filtrate from an Ewald test meal gave the biuret reaction. This conclusion is based upon the assumption that the biuret reaction is an indication of the presence of peptones. These gastric juices, although they give no evidence of presence of HCl to the ordi-

nary color tests (Acid indicators), showed a feeble dissociation of methyl acetate by the Hoffmann-Ostwald method of determining the free HCl. This probably indicates that the HCl may be so loosely combined with proteoses and acid albumen, that it can readily dissociate and in the presence of pepsin produce peptones, although the ordinary color tests — congo red tropaeolin 00, phloroglucin-vanillin —, are negative.

We had in these persons a demonstration of gastric juices that (1) contained no free HCl, but combined HCl, because they were capable of producing biuret reaction; (2) could not coagulate milk; (3) they gave the biuret reaction with an Ewald test meal. The conclusion seems justificable that pepsin was present, but rennin or chymosin was absent, which is difficult to harmonize with the view of Pawlow that the proteolytic an the milk curdling effects are due to one and the same molecule.

Schlussätze zu obiger Arbeit.

Zu Gunsten seiner geistreichen Hypothese, dass die proteolytische und milchgerinnende Wirkung verschiedener Verdauungssäfte nicht verschiedenen, sondern ein und demselben Molekül angehören, führt der hochverdiente Forscher (Pawlow) weiter an (l. c. p. 449):

I. Dass die Milchkoagulation eine Komplikation des Eiweissmoleküles sei, ganz entsprechend der Vereinfachung desselben, welche unter Einwirkung der proteolytischen Fermente, wie Pepsin, stattfindet und citiert die Untersuchungen von A. Danilewsky und Okunew, nach welchen Koagulation einiger Albumosen durch Labfermente nachgewiesen worden seien.

Dass zur Demonstration einer solchen Annahme vorerst die Darstellung eines reinen Labfermentes nötig ist., d. h. ohne die Wirkung eines Eiweissferments zugleich zu besitzen, ist evident. Aber in jener ganzen Arbeit ist doch das Hauptargument, dass solche exklusiv Labwirkung aufweisende Enzyme nicht vorkommen.

Die reversible Wirkung von einem Fermente ist zuerst von Loewenhardt und Kastle (American Chemical Journal) nachgewiesen worden und zwar die zerspaltende und zugleich synthetische Wirkung der Lipase auf das Athylum butyricum. Mit solchem *relativ einfachen* Körper (substrat) arbeitend, lässt sich einwands*frei bei der Lipase* zugleich analytische und synthetische Wirkung *nachweisen.*

Beweisen aber die interessanten Arbeiten von Danilewsky und Okunew in der Tat, dass eine Koagulation von Albumosen durch reines Labferment vorkommt? Ist es nicht eine Präzipitation und keine Koagulation? Wie man analog ein Präzipitin streng von einem Koagulin zu unterscheiden hat.

Auffallend ist es, dass, wenn den proteolytischen Enzymen der Verdauungssäfte zugleich auch koagulierende Eigenschaften zukommen sollen, diese letztere Wirkung sich nur an einem Proteid demonstrieren lässt (der Milch, nämlich Kasein). Wenn das eine allgemeine Qualität solcher Fermente wäre, müsste es sich doch auch an anderen Eiweisskörpern demonstrieren lassen. Um reversible Effekte eines Fermentes zu demonstrieren, müssen beide Wirkungen an ein und demselben Proteid bewiesen werden. In dem Falle der Verdauungssäfte scheitert diese Bedingung, denn das Pepsin kann man nur durch seine analytische Wirkung auf Eiweiss beurteilen (Mett'sche Methode), die hypothetische synthetische Wirkung jedoch nur auf das Proteid der Milch.

II. Eine weitere Begründung ihrer Annahme suchen die Herren Professor Pawlow und Dr. Parastchuk darin, dass Labferment bei verschiedenen Tieren und Pflanzen vorkommt, wo es niemals mit Milch in Berührung kommt, und erheben die Frage: Wozu?

Hier wird nun der Zweckbegriff in die Diskussion eingeführt und als Argument benutzt. Wenn in der Tat Lab bei Tieren und Pflanzen vorkommt, wo es niemals mit Milch in Berührung kommt, so ist die Frage: Wozu? schwer zu beantworten. Die genannten Forscher wollen zu verstehen geben, dass eben Chymosin und Pepsin identisch sind (ist Pepsin vorhanden, dann ist Labwirkung ebenfalls nachzuweisen). Nun gibt es aber auch proteolytische Enzyme in Pflanzen, wo es niemals mit zu verdauendem Proteid in Berührung kommt, in dem Saft der reifen Ananas zum Beispiel. Ich habe mit dem Saft der Ananas gearbeitet und einer meiner Assistenten mit dem Saft der Suavafrucht. Obschon beide Säfte Milch koagulieren (ein Tropfen Ananassaft bringt 20 ccm Kuhmilch in 15 Minuten zur Gerinnung), konnten wir uns nicht überzeugen, dass diese Wirkung von einem Ferment abhing. Denn erstens waren beide Fruchtsäfte intensiv sauer, und neutralisierten wir die Säure, dann hoben wir die Labwirkung auf. Und zweitens vertrugen die Säfte einen höheren Grad der Erhitzung (70° C.) als dem Lab für gewöhnlich d. h. ohne Zerstörung zuträglich, zugeschrieben wird. Pawlow und Parastschuk würden vielleicht den Verlust der Milchge-

rinnung bei der Neutralisation einer Hemmung des Labs durch die angewandte Decinormalllösung von NaOH oder KOH zuschreiben oder einer Erhöhung der Salzkonzentration, doch konnte eine darauf folgende Verdünnung die Labwirkung nicht wieder herstellen.

Eine sehr interessante Stütze der Ansicht, dass Pepsin und Chymosin nicht identisch, sondern zwei spezifische Fermente sind, liesse sich auf biochemischem Wege erzielen. Es ist vielleicht nicht logisch, durch Untersuchungen, die noch nicht vollendet sind, etwas beweisen zu wollen. Doch kann ich als vorläufige Mitteilung nicht unterdrücken, mitzuteilen, dass das auf biochemischem Wege von Serum gewonnene Antilab nicht auch zugleich ein Antipepsin ist, was doch der Fall sein müsste, wenn das dem Tier injizirte Pepsin als Ferment ein chemisch einheitliches Molekül darstellte.

Auf biochemischem Wege steht auch die Vermutung nahe, dass die Labfermente der Säugetiere verschieden sind, denn ein Antilab durch Injektion des aus Kälbermagen gewonnenen Ferments (Methode nach Ivar Bang) erzielt, hebt die milchkoagulierende Wirkung von Hundelab nicht auf.

Ich habe bisher nur mit den Enzymen aus Magen von Hunden, Meerschweinchen, Katzen, Kälbern und Schweinen gearbeitet und sie mit den Enzymen aus menschlichen Magensäften verglichen und habe den Eindruck gewonnen, dass das Lab eines jedes dieser Tiere spezifisch am effektivsten auf die Milch desselben Tieres koagulierend wirkt.

Hat man aus Meerschweinchenserum ein Antilab gewonnen, welches durch Injektion von Kälberlab erzeugt wurde, so ist die hemmende Wirkung des Antilabs am effektivsten mit der Kuhmilch zu demonstrieren, d. h. mit der Milch der Tiergattung, von welcher das Lab ursprünglich genommen wurde. Diese feinen Unterschiede berechtigen zu der Ansicht, dass „reciproke“ biochemische Beziehungen zwischen den verschiedenen Labarten und Milcharten bestehen.

Alle diese Beobachtungen sollen jedoch nicht als Ablehnung der herrlichen Arbeiten Pawlow's gelten, sondern nur betonen, dass die vielbesprochene Identität des Pepsins und Labs noch nicht endgiltig bewiesen ist. Das Denken dieses geistreichen Physiologen hat bisher so wunderbar befruchtend gewirkt, dass sicher weitere wertvolle Beiträge von ihm zu erhoffen sind.

Druck von L. Schumacher in Berlin N. 24.

DISCUSSION.

DR. J. KAUFMANN, of New York City: I think a good opportunity for studying this question of whether or not there are two different enzymes in the stomach offers itself in those cases where the gastric secretion gradually disappears, as in carcinoma. I found in one case of carcinoma, after partial resection of the stomach, the pepsin test positive, the rennet test negative. In another case of carcinoma with pyloric stenosis, both tests were at first negative. Then a gastro-enterostomy was done and the patient rapidly gained about thirty pounds in weight. Both tests then gave positive results. Subsequently, a partial resection was performed, and after the second operation the pepsin test was negative and the rennet test positive. The results of the tests in these cases would speak for the presence of two different enzymes in the gastric juice if such clinical tests are reliable. In all cases of achylia gastrica which I examined, both tests were regularly negative.

DR. LAFAYETTE B. MENDEL, of New Haven, Conn.: My impressions on this subject have been gained from the papers that I have read, as I have had no personal experience. From what I have read, I have never been convinced of the presence of different rennin-like enzymes in the gastric juice; and I think it is rather surprising that in all these years the claims of those who hold that view have not been verified. I would like to say, however, that it was not Pawlow who first advanced the idea that rennin and pepsin are one and the same, but really the brilliant Nencki.

DR. EINHORN: While I have had no personal experience along this line, I have met with cases where I could not demonstrate the presence of pepsin but still rennin was present. I have never seen pepsin without rennin. The lack of uniformity in our results in this work may be due to our faulty methods of examination.

DR. A. L. BENEDICT: I have noticed that certain samples of milk will not react to the rennin test, and I have seen several samples of stomach contents in which I got the reaction for rennin, but not for pepsin, or vice versa. I do not know the cause for this, but it suggests the existence of separate ferments, as is commonly held to be the case.

DR. MENDEL: The failure to react was perhaps due to the presence of formaldehyde in the milk, this could be proved by testing for formaldehyde.

DR. BENEDICT: That might be the explanation in some cases, but one of my cases was seen before formaldehyde was on the market.

[Reprinted from *American Medicine*, Vol. X, No. 13, pages 520-525, September 23, 1905.]

DIVERTICULUMS OF THE ESOPHAGUS, WITH REPORT OF CASES.[1]

BY

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Ectasies or enlargements of the esophagus are of two kinds, entirely distinct in their causation and pathology, namely: (1) Dilation or diffuse enlargements involving more or less the entire periphery of the tube and due usually to organic or spasmodic stenosis; and (2) diverticulums, localized bulgings or saccular protrusions originating from a circumscribed portion of the wall of the esophagus.

Systematic knowledge of diverticulums of the esophagus dates from the classic article of Zender and von Ziemssen.[2] Since then numerous papers have appeared, embodying reports of cases, etiologic, and pathologic studies, methods of diagnosis and treatment, etc. The most complete and most comprehensive work on the subject is the 206-page monograph by Dr. Hugo Starck, "Die Divertikel der Speiserohre," Leipzig, 1900. Diverticulums of the esophagus are divisible into two varieties sharply distinct in their origin and course—pressure diverticulums and traction diverticulums.

Pressure diverticulums are those produced by the esophageal pressure, involved in swallowing, acting upon a circumscribed area of weakening in the esophageal wall. They are uncommon, and occur less frequently than traction diverticulums. They may be single or double. They are rounded or oval saccular protrusions communicating, by a circumscribed round or slit-like opening, with the lumen of the esophagus. In size they are from insignificant bulgings to large cavities with a capacity of several hundred cubic centimeters. Their walls vary in structure in different cases. Occasionally there may be a fairly complete muscular layer throughout. In some cases the muscular layer is thinned,

[1] Read before the eighth annual meeting of the American Gastroenterological Association, held at the Academy of Medicine, New York City, April 24 and 25, 1905.

[2] Vol. viii of von Ziemssen's Cyclopædia of Practical Medicine, 1878.

sparse, partial or interrupted. In most cases, however, muscular tissue is entirely absent from the diverticulums, the wall being formed of a hernia-like bulging of the mucous membrane through a cleft or defect in the muscular wall of the esophagus. The esophageal diverticulums, that give rise to perceptible clinical symptoms and come under observation and treatment, are practically all of the pressure variety. The determining factor in the formation of pressure diverticulums is the existence of a weak place or defect in the wall of the esophagus, which in the course of time is converted into a protrusion by the pressure exerted during deglutition. Various explanations have been offered as to the original causation of the weak places in the gullet wall. The theories offered may in general be divided into two classes: 1. Those assigning a congenital or developmental origin. 2. The mechanical, traumatic and inflammatory hypotheses. The etiology is still under discussion, awaiting final settlement. It is quite possible that in different cases different primary etiologic factors are involved.

According to the congenital theories, the diverticulums originate in points where there has been a defect or abnormality of development, as at the middle of the esophagus, where this canal is finally separated from the respiratory passages, or in clefts in the upper part of the esophagus, or in pouches in the pharyngeal region, similar to pouches which are normal to some species of animals. The general trend of opinion seems to be rather opposed to the genetic theory of diverticulum formation except, perhaps, in some of the diverticulums that develop entirely in the vicinity of the bifurcation of the trachea.

According to the mechanical theory, the points of weakness that serve as starting points for the formation of the diverticulums are produced by traumatic, inflammatory or cicatricial conditions. These may act, either by causing a diastasis or splitting of the muscle fibers of the esophageal wall, through which a hernial protrusion of the mucous membrane takes place, or by causing an area of weakness followed by a general bulging involving the muscular as well as the other tunics. One of the most common causes alleged is the lodgment of solid bodies in the esophagus such as to produce diastasis, or traumatic or inflammatory weakening. The lodgment of a mass of very hot food, with tissue injury from the heat, has been assigned as a cause in at least one case. Localized inflammations from any cause are supposed to

be capable of producing the determining weakness. Ulcers, as from swallowing caustic substances may directly produce areas of weakening, or be followed by cicatrices which, instead of contracting and causing stenosis, may become thinned and stretched and weakened so as to yield to the internal pressure in the same manner as a ventral hernia may result in the cicatrices of abdominal operations. Wounds and injuries, as from the use of sounds, may result similarly. Stenosis may, at times, be influential in the production of diverticulums, though contractions of the esophagus are more apt to be secondary to diverticulum formation. Finally, preexisting traction diverticulums may serve as starting points for the development of pressure diverticulums.

The esophagus in its course presents three points of narrowing, one just below the cricoid cartilage, one just above the cardia and one in the middle of the esophagus near the bifurcation of the trachea. These points of narrowing are supposed to have special bearings on the formation of diverticulums, since at these points objects are most likely to lodge, injuries are more apt to occur, and the esophageal pressure is the greatest.

With regard to location, while pressure diverticulums may occur in any part of the esophagus, they are divisible into two wellmarked classes: (1) Those at the junction of the pharynx and esophagus; (2) those below the cricoid cartilage. The most frequent site of diverticulums is the pharyngoesophagus, at the level of the cricoid, and proceeding from the posterior or lateral aspect of the wall. In 99 out of 121 cases, or 82%, which Stárck was able to collect in 1900, the site was at this point. There are two anatomic factors that are regarded as especially favoring the formation of diverticulums in this region, one is, that just below the cricoid is situated the most constricted portion of the esophagus; the other, that there is an area of muscular weakness posteriorly and laterally opposite the cricoid, the fibers of the constrictor muscles here running transversely only, without any longitudinal fibers to give additional support, so that traumatic diastasis of the transverse fibers is more easily accomplished. Males are overwhelmingly more subject to this variety of diverticulums than are females. In 86 of Starck's cases, 67 were in males and 19 in females. The trouble develops in the later years of life, especially in the period from 40 to 60. Only five patients were under 30. Esophageal pressure diverticulums proper are those situated below the region of the

cricoid. They may rise from any level of the gullet, but most often rise in the middle third near the bifurcation of the trachea, and oftener in the lower than the upper third (Starck). They proceed from the anterior or lateral aspects of the esophagus, not from the posterior. They are the least common of all the varieties of diverticulums of the esophagus. Starck, in 1900, collected only 20 cases. The later years of life (after 30), are more prone to the incidence of the disease, though cases have been observed at the ages of 3, 6, 18 and 21. The two sexes are nearly equally affected—14 males and 11 females in Starck's series.

Traction diverticulums are pouches or eversions produced by traction from without, or cicatrices or bands adherent to the esophageal wall. They are the commonest form of the esophageal diverticulums, occurring, it is said, in from .5% to 4% of all adults. In shape they are conical and pointed, the apex being the point where the traction is exerted. They are small in size, ranging up to 20 mm. in length, and 3 mm. to 18 mm. in diameter. They are usually directed upward from the point of entrance into the esophagus, sometimes stand at right angles with it, least frequently are directed downward. In the great majority (nearly four-fifths) of the cases the diverticulums occur singly; in about a seventh they are double; occasionally three or four, or even five or six diverticulums are present in the same esophagus. The walls of the diverticulums may consist of mucous membrane only, or there may be more or less complete muscular coat also; cicatricial fibrous tissue may also be present. Males are more subject to the condition than females. Of 164 cases, 95 were in males and 69 in females.

Traction diverticulums are situated nearly always at the middle of the esophagus, in the vicinity of the bifurcation of the trachea, with some preference for the right side. The great majority of them result as a sequel of inflammation of the tuberculous, anthracic, or other condition of the bronchial glands situated at the bifurcation of the trachea. The cicatricial contraction following such inflammation, through adhesions to the esophageal wall, produces traction diverticulums of the latter. Pulmonary lesions, tuberculous, interstitial pneumonia, pleural or pericardial inflammation, or other local conditions, involving esophageal adhesions and contractions, may in a similar manner produce traction diverticulums. A developmental origin for these

diverticulums seems also to have been demonstrated in a few of the cases; the final embryonic separation of the esophagus from the respiratory passages takes place near the bifurcation of the trachea, and the persistence of genetic adhesions or bands between the gullet, and the trachea may determine the formation of traction diverticulums at this point.

On account of their symptomless development, the age of incidence of traction diverticulums has not been definitely determined. It has been suggested that as tuberculosis of the bronchial glands is a disease of early life, these diverticulums probably develop in the young, but at autopsy a majority of the cases are found in subjects over 30; a few cases having been observed from 1¼ to 10 years, and many cases from 10 to 30.

On account of their small size, traction diverticulums produce no manifest symptoms and rarely if ever have they been diagnosticated or even suspected during life; they are first found at autopsy. However, while ordinarily harmless and symptomless, they are subject to possible accidents and conditions that may lead to grave results. The principal danger is that of perforation of the diverticulum, which is said to occur in 10% of the cases. This accident may be brought about in various ways, as by lodgment of foreign bodies causing direct rupture (fishbones) including inflammation and ulceration; or by the lodgment and stagnation of food causing infection and setting up an inflammation. The perforation may take place in the trachea or bronchi, pulmonary cavities, the mediastinum, pericardium, etc., with consequent infections and inflammatory conditions, as local abscesses, pulmonary gangrene, pleurisy, empyema, pericarditis, pneumopericardium, etc. These diverticulums may form starting points for carcinoma or tuberculosis; also for the formation of pressure diverticulums.

Mixed and False Diverticulums.—Cases of mixed diverticulums have been observed in which pouches obviously primarily produced by traction had undergone enlargement by pressure within the esophagus. Such cases arouse the suspicion that pressure diverticulums of the middle portion of the esophagus may similarly start from traction diverticulums, the second enlargement of the sac effacing the evidences of the primary traction causation.

Abscess cavities and similar ulcerated spaces in adjacent organs, ulcerating into, and forming a communica-

tion with the esophagus, such as pulmonary cavities, cancerous ulcerations, retropharyngeal abscesses, etc., present conditions simulating the diverticulums. Such form the class of false diverticulums.

Symptoms and Clinical Manifestations.—Small diverticulums run a symptomless course, with no suspicion of their presence, until discovered at autopsy. Large pressure diverticulums exhibit serious clinical manifestations and may come under the attention of the physician. Their development and course are gradual and protracted, extending over months or years.

The initial symptoms observed in the upper diverticulums, aside from whatever history there may be of the lodgment of foreign bodies, etc., are those of irritation, expectoration of mucus, irritable cough, hawking, or a slight difficulty in swallowing. In the diverticulums situated lower, the earliest symptoms are usually a sense of dull, vague pressure, and a dull pain or burning during swallowing. In the fully developed condition, difficulty in swallowing is one of the chief symptoms. The dysphagia is caused by the food passing into the diverticulum; by the fact that the diverticulum, when filled and distended by the food, may, by pressure against or distortion of the esophagus, obstruct the passage of food into the stomach; or by occasional development through nonuse, of contraction or stenosis of the esophagus below the mouth of the diverticulum. The patient feels the food lodge or go astray; he may be able to force it down only with great difficulty or effort; or may become quite unable to swallow certain kinds of food. Usually solid foods or large masses give the most trouble; exceptionally fluids are swallowed with greater difficulty than solids. The dysphagia may be constant and occur at every attempt at eating; or, especially in the early stages, it may be occasional, intermittent, occurring only at the beginning, or at the end of the meal.

Regurgitation of food is an important symptom, the necessary consequence of the failure to force swallowed foods into the stomach. Regurgitation may take place immediately after swallowing the food; or it may be delayed for a variable period up to one or even to 24 hours. In some cases it is accomplished with great ease and no effort, often, abruptly and unexpectedly; in other cases there is violent effort and straining, as in vomiting. Some patients acquire the ability to empty the filled diverticulums at will; in some it is brought about by changing the position of the body.

The character of the regurgitated material varies according to the length of time it has been retained in the esophagus. Food immediately regurgitated returns unchanged. If it remains in the diverticulum for a sufficiently long time, bacterial growth occurs, causing fermentation, etc. Salivary digestion only can take place. Mucus, sometimes in large quantities may be mixed with the food material. The specific gastric elements, hydrochloric acid, rennet, etc., will necessarily be absent from material that has never reached the stomach, and the determination as to the presence or the absence of these elements will have an important bearing on the diagnosis. The stagnation and fermentation of the food in the diverticulums is apt to produce inflammation and ulceration of their lining tissue. In the high diverticulums, the odor from the stagnant food is a common and disagreeable symptom.

Pain from distention of the diverticulums is a common symptom, especially in those situated low down. In pharyngoesophageal diverticulums, pain is sometimes absent. It arises from the distention and dragging of a heavy, filled sac, irritation of ulcers or inflamed areas in it, or pressure on neighboring organs. The pain may be sharp and severe, or there may be a dull sense of weight and pressure. Pressure of the distended diverticulums on the heart may cause palpitation; on the trachea or bronchi, dyspnea; on the cervical bloodvessels, cephalic congestion; on the laryngeal nerves, apnea, etc.

Diverticulums situated in the cervical region may be manifested by a tumor, visible and palpable in the neck region, appearing or disappearing according as the sac is full or empty. In many such cases this is a conspicuous symptom, yet it may be absent with quite a large diverticulum. Peculiar sounds of a murmuring, gurgling, rattling or bubbling character, occurring on swallowing fluids or solids, on the entrance of air, or in changing position, are common symptoms, especially in the high diverticulums. These sounds are due partially to the movement of air bubbles associated with the food matter.

In the diverticulums situated at the upper limit of the esophagus, the proximity of the seat of dysphagia to the glottis gives the tendency and danger of food matter being inspired into the respiratory tract. Many patients are compelled to exercise constant care to prevent this accident, the slightest carelessness or forgetfulness often resulting in inspiration of food, followed by violent

coughing paroxysms to expel it. One of the commonest terminal complications of this form of diverticulum, is pneumonia and pulmonary gangrene, caused by inspiration of foreign matter into the respiratory passages; this, especially in patients suffering from malnutrition, sets up infectious pulmonary processes that prove fatal.

With impairment of the power of swallowing, and failure of food to reach the stomach, conditions of starvation, malnutrition, and emaciation are occasioned, which constitute the most serious aspect of esophageal diverticulums, and the greatest menace to health and life. Starvation symptoms appear in a large portion of the cases, and emaciation and asthenia may reach the most extreme degree. Death from starvation has been the most common cause of death in the recorded cases, and is the outcome the patient must expect unless relieved by treatment.

Physical Examination.—In the diagnosis of diverticulums of the esophagus, the aim is, not only to determine the existence of diverticulums, and to differentiate them from diffuse dilation, stricture, carcinoma, etc., but also, to determine their number, position, size, condition of the sac (inflammatory processes, etc.), and the patency of the esophagus below the sac. Exploration by means of the esophageal sound or stomach-tube reveals much important information. By the sensation imparted to the fingers when the point of the tube enters the diverticulum, the position, direction and distance of the opening from the teeth can be judged. By similar sensations, the contact of the end of the tube with the bottom of the sac can be judged, and the depth of the diverticulum thus determined. The capacity of the sac can be measured by the amount of fluid that can be introduced into, and withdrawn from it. By filling the sac with fluid that will stain or mark the tube, its depth can be determined, by the distance to which the tube is stained. The patency and passability of the esophagus below the diverticulums should also be determined by the sound or tube, entry into the stomach being demonstrated by the withdrawal of fluid containing specific gastric elements (hydrochloric acid, rennet, etc.); by palpation in the epigastrium; by forcing air into the stomach; auscultating over the cardia during the act of swallowing. By percussion over the seat of the diverticulum during the act of drinking liquids, the area of dulness may be accurately determined.

Two tubes may be passed simultaneously; after one

tube is passed into the diverticulum, a second tube is passed alongside and so guided into the stomach or into a second diverticulum. By the introduction of two tubes into different cavities they may with certainty be explored separately, and their independence of each other demonstrated.

By having one tube fenestrated in the Rumpel double-tube test, it can be determined whether the enlargement of the esophagus is a circumscribed diverticulum or a diffused dilation. A stomach-tube, with opening along its sides for a distance of 14 inches, is introduced into the stomach; that it has entered the stomach is to be demonstrated by the character of the fluid withdrawn through it or through a smaller tube passed through it for the purpose. A second tube is introduced alongside the first into the esophageal ectasis. Fluid colored with eosin, cochineal, or other distinctive matter is poured through the esophageal tube into the sac. If the esophageal enlargement is a diffuse enlargement, some of the fluid will escape through the lateral openings into the stomach, and will flow out through the smaller stomach-tube. If the fluid does not escape from the smaller stomach-tube, it shows the enlargement is a circumscribed diverticulum, the walls of the esophagus being in close contact with the fenestrated tube so as to prevent escape of the colored water.

Determination of the clinical characters of material regurgitated, or removed from the cavities involved, is important in diagnosis. The contents of a diverticulum, or of a diffuse esophageal dilation, is composed of mucus and food particles in varying amount, with at times traces of blood, but with no hydrochloric acid or ferment. In rare instances, traces of hydrochloric acid, rennet, and pepsin may be found in those cases, in which there is hyperchlorhydria with regurgitation of fluid from the stomach, which passes into the sac. Inspection and palpation may reveal evidences of tumor, varying according to the fulness or emptiness of the sac, in diverticulums in the cervical region. By auscultation of the deglutition sounds, abnormal sounds caused by the diverticulum formation, and perhaps, absence of the swallowing sounds at the cardiac orifice, may be detected that will be of diagnostic value. By percussion, comparing results when the sac is full and when it is empty, the diverticulum can sometimes be outlined. A pathognomonic sign is the appearance, and gradual decrease of a circumscribed area of dulness over the site of the divertic-

ulum while fluid is being introduced into it; and the gradual decrease in this area of dulness when the individual reclines toward the opposite side, showing the gradual emptying of the sac. The use of the esophagoscope may show the opening into the sac and yield information as to the presence of inflammation, ulceration, etc., in the surface of the parts involved. Transillumination, by the introduction of an electric light into the diverticulum, may show its position and extent, although as yet this method of examination has not been of much service. The diverticulum may be filled with a bismuth mixture, and then subjected to a röntgen-ray examination, which may afford some information as to its locality, shape, and size.

Treatment.—In accessible esophageal diverticulums in the cervical region, surgical procedures for radical cure are available. Starck, in 1900, collected 20 operative cases. In 18 of these cases the sac was resected, with a mortality of 5, one from aspiration pneumonia, two from secondary hemorrhage, two from cervical phlegmon. In the later cases, the mortality was far less than in the earlier cases, so there is reason to believe with perfection of technic, the mortality may be brought to a lower figure. In two of the cases, the sac was obliterated by invagination, with successful outcome. The establishment of a fistula from the borders of the diverticulum of the skin, to afford drainage for the sac, has also been proposed, and once done (in this case death resulted from pneumonia).

In nonsurgical cases, the conditions may be palliated, by keeping the diverticulum empty, and cleansed, by irrigation. Astringent or antiseptic applications, or irrigations may also be employed to relieve inflammation and ulcerative conditions in the sac. By relieving the sac of irritation and distention, its conditions may be much improved, and it may undergo material decrease in size. While amelioration of the trouble may result from these measures, radical relief cannot often thus be counted on. As the greatest trouble in these cases arises from starvation, so the greatest results of treatment, often amounting to symptomatic cure, may be obtained from proper nutrition. When dysphagia is only moderate, a sufficient amount of food can sometimes be introduced into the stomach by natural deglutition; by careful selection of easily swallowed foods; careful eating; and by keeping the esophageal passage open and straight by the sound, or bougie. In cases, in which

the power of swallowing is greatly impaired or entirely abolished, gastrostomy and feeding through the fistula affords one method of keeping up nutrition. It has been performed in a few instances, but usually, probably on account of the enfeebled condition of the patient when undertaken, with immediately fatal results. In cases in which the esophagus is entirely impermeable, gastrostomy may afford the only means of maintaining nutrition, beside affording an opportunity for retrograde sounding. When dysphagia is extreme, and the esophagus passable by the stomach-tube, a far better method of feeding is by gavage, which the patient can learn to perform for himself. An entirely adequate amount of food can thus be introduced into the stomach without excessive discomfort, through the natural passages instead of a troublesome artificial fistula, and without the necessity of a formidable operation. While this method of feeding is less pleasant, and less sociable than the natural method, yet it affords a means of comparative cure, restores the normal nutrition and strength, and is the alternative to death from starvation.

Tube feeding.—The method of tube feeding which I teach my patients to employ is as follows:

A large-caliber, soft-rubber stomach-tube having fairly thick walls, with two end and two side openings, and measuring about 36 inches in length, is selected. When there is difficulty encountered in passing the tube by the diverticulum, I have a hard-rubber, olive-shaped tip about a half inch long made so as to be securely fastened to the proximal end opening, which greatly facilitates the introduction of the tube. The tube is put into warm water and inserted down, but not through the cardiac orifice. The esophagus and diverticulum are carefully and thoroughly cleansed with decinormal warm salt solution, after which the tube is pushed into the stomach. To the distal end is attached a glass funnel having a capacity of about one pint. Food is then poured into the funnel and through the tube into the stomach, and the meal is finished with about six ounces of warm water. Care is taken to withdraw the tube before the water has entirely passed out in order to prevent any food being drawn up with the tube into the esophagus.

The following are some of the foods included in the dietary: Beef juice, rich soups and broths, including those containing cream, eggs and wine; puree of all vegetables which do not have tough fiber, among them being puree of spinach, beans, peas, celery, asparagus and potatoes; milk and cream; cocoa, chocolate, coffee, tea and some wines; eggs, raw or soft; scraped meat and fish chowder; porridge made from thoroughly cooked breakfast cereals; a few sweets such as bees' honey, calf's foot jelly, and simple custards. Once a week the individ-

ual is instructed to masticate thoroughly several mouthfuls of some hard substance like crackers with butter, or bread with butter, before one or other of the meals, and even to swallow some portion of it in order to keep up the secretion of saliva in the mouth. By immediately washing out the diverticulum and esophagus no harm is done.

CASE I.—S. T. F., aged 45, married. At the age of 7 he had a severe attack of typhoid fever, and double pneumonia. At 12 he had a severe attack of bloody flux, which nearly proved fatal. He had several attacks of grip. A maternal aunt had for years suffered from inability to swallow, and at autopsy a tumor was found at the cardiac orifice with dilation of the esophagus above.

Patient's present illness began in 1898. While at dinner, the attack began abruptly with sharp agonizing pains beneath the ensiform cartilage. It lasted a few minutes and then passed off. Later in the night, he had another attack of pain, for which a hypodermic of morphin was necessary. The following morning he experienced great difficulty in forcing food into the stomach. The attacks of pain, coming on in the evening, continued to recur almost every evening up to July 1, 1900, since which time they have come less often, and with less severity. The difficulty in forcing food into the stomach gradually increased until he became unable to take any solid food, living entirely upon milk and thin soups. Since October 1899, he had been steadily losing flesh until at the time of coming under treatment his weight was 40 pounds below normal. He consulted me September 17, 1900, for difficulty in swallowing, at that time showing the following condition: Bowels constipated; appetite good; tongue clean; skin and sclerotics clear; color good; breath not offensive. Heart, lungs, liver and spleen apparently normal; panniculus good. Belly normally formed. Splashing sound elicited only over upper half of epigastrium. No pain or tenderness elicited on deep pressure over stomach. One hour after test-breakfast the gastric contents showed hydrochloric acid present; total acidity 42; blood, lactic acid, mucus, pathogenic bacteria absent. On passing the stomach-tube, when the tip was at a distance of 13 inches from the incisors, it was felt to glide to the right; it lodged and could not be passed further. A second tube, passed while the first remained in the esophagus, sometimes slipped easily into the stomach, and sometimes turned to the left and lodged, as in the case of the first tube. I introduced into the stomach a tube having numerous side perforations extending upward from its tip for a distance of 10 inches, and through this tube was passed a smaller tube. An ordinary stomach-tube was introduced into the right sac, and ten ounces of colored liquid were poured through it, distending the sac. Not any of the colored liquid passed into the stomach and all of it came out through the esophageal tube, proving we were dealing with a true diverticulum. Upon introducing the esophagoscope, the interior of the esophagus was examined with difficulty. At the bottom of the right diverticulum was a bright red spot about an eighth inch in diameter, which was probably an ulcer. When the right diverticulum was filled with water, an area of dulness was easily made out on percussion to the right of the lower end of the sternum, which gradually disappeared when

the patient reclined upon his left side. A No. 38 French esophageal bougie passed easily into the stomach with but slight resistance at the cardia. One hour after eating, a plateful of rolled oats was nearly all washed out of the diverticulum, unchanged and without a trace of acidity in it. Upon washing out the stomach immediately afterward only a small amount was found.

Subjective Symptoms.—He experienced difficulty in swallowing food; it seeming to lodge somewhere above the cardia, caused a fulness and sense of constriction, together with considerable pain and discomfort, which at first could be relieved by gulping a large swallow of water.

Treatment.—The patient was put upon a semisolid diet consisting of soft eggs, rich soups, coffee, cocoa and bees' honey. The only medication ordered was sodium bicarbonate an hour after meals, and .02 gm. (¼ gr.) doses of codein whenever necessary to control the pain. After each meal the patient was instructed to wash out the diverticulum with warm normal salt solution. This line of treatment was continued, with slight variations from time to time, until December 7, 1902, upon which date the patient weighed 169½ pounds, a gain of about 8 pounds in two years. In the two years during which he was under observation, he had frequent attacks of pain, while the difficulty in forcing foods into the stomach remained as it was in the beginning. He had a keen appetite and enjoyed his food. Bowels were constipated, requiring the use of oil enemas. At this time I advised him to take all food and liquid through a No. 38 French stomach-tube, feeding himself three times a day at the usual meal hours. After a few days he became quite expert in the gavage. On January 2, 1905, he came again to see me. Since he began the gavage he has practised it three times a day continuously with the exception that one meal a month he has attempted to take without the tube in order to find out if the difficulty was becoming less. At the present time all sense of lodgment of food on the left side has entirely disappeared and he has much less difficulty in forcing food into the stomach. He has not experienced any pain for many months past. He has no trouble from constipation since he has been able to eat fruit. He weighs 189 pounds, a gain of 20 pounds since he began gavage. He feels strong and vigorous and is the picture of sound health.

CASE II.—On October 20, 1904, Mrs. I. S., aged 60, was referred to me by her laryngologist, with a history of progressively increasing dysphagia. She had been all her life a hard working woman, and had had good health up to three and a half years before, when the sudden death of a near relative brought on severe nervous symptoms, together with a severe attack of nervous dyspepsia. Shortly after this she began to notice that when she was very nervous she had some difficulty in swallowing foods. This had grown progressively worse, until at the time I first saw her she was only able to swallow bland liquids. She had lost about 40 pounds, although her general appearance did not suggest serious illness, inasmuch as her present weight was not below the standard for her height. Heart, lungs, liver, and spleen were apparently normal. Examination of blood showed a slight degree of chloranemia, with a very slight increase in the leukocytes. She experienced no pain at any time in any place. Appetite was so keen as to become annoying, since she was not able to satisfy it with solid food. Bowels were constipated. The patient was so very nervous and apprehensive, that it was

with difficulty any sort of an examination was carried out. On attempting to introduce a No. 34 French stomach-tube, the tip lodged in a pocket to the right of the median line, when a distance of nine inches from the incisors was reached. A second tube, passed in beside the first, lodged in a similar manner, and could not be made to enter the stomach. After several trials with a No. 32 olive-tipped silkwormgut esophageal tube, it passed easily into the stomach, and a few cubic centimeters of acid chyme were withdrawn. Eight ounces of water were poured through the tube, which had lodged in the diverticulum, and the entire amount, unchanged, was recovered by siphonage. As the patient refused to submit to further examination, and rejected the suggestion of gavage, nothing further was attempted. On January 24, 1905, her son-in-law reported the patient's condition to be about the same as when I saw her last, and so far as he could see she had not failed in either strength or weight during the intervening period, although she had lived entirely upon milk and soup.

CASE III.—On November 16, 1904, I was called in consultation with Dr. J. R. Bromwell of this city, to see Miss C. L. G., who had been suffering with dysphagia since February, 1903. She had scarlatina at the age of 5 and measles at 7. Menstruation began at 12 and ceased at 50, about the time dysphagia appeared. She had suffered from constipation all her life. For the past 12 years she was subject to violent, but brief attacks of cramps in the right hypochondriun. Ten years ago she had an attack of "bilious colic," so severe as to raise a question of the presence of gallstones. For most of her life, she had been subject to violent headaches, less so in recent years. She had whoopingcough at 43. Three years ago a swelling developed in the right groin, probably a hernia. When her symptoms first made their appearance, the attacks were intermittent and were considered, and probably were, due to nervous dyspepsia. Under appropriate treatment, and with a sojourn at a health resort, the patient improved rapidly and gained in weight, having had no symptoms for some months. During the past few months, however, dysphagia has returned, and increased steadily, until now she is unable to force solid food into the stomach. During the past few months, she has lost from 15 to 20 pounds, and looks ill. Heart, lungs, liver and spleen are apparently normal. Urine and feces are normal. Examination of blood shows a moderate degree of anemia, but no increase in the leukocytes. She complains of the usual symptoms of dysphagia, together with a severe pain located behind the ensiform cartilage, which, while it lasts, is so severe as to make her unable to walk, but which immediately passes off after she is able to regurgitate food from the esophagus. Except for dysphagia and the pain coming on at meal times, she has no pain or discomfort. Appetite is good. Bowels have been obstinately constipated. Upon introducing a 34 French tube when the tip reached a point of 12 inches from the incisors, it was distinctly felt to glide to the right and lodged in a pocket. A second tube passed in beside the first sometimes lodged in a pocket at a distance of 14 inches from the incisors to the left, and sometimes slipped easily into the stomach. Rumpel's test with colored liquids was carried out and a right diverticulum found which held about 7 ounces and a left diverticulum which held about 4 ounces of liquid. When the liquid was introduced gradually into the right diverticulum an increasing area of dulness on percussion was readily made out at the

lower end of the sternum, which gradually diminished when the patient was turned upon her left side. When the sacs were carefully filled with eosin colored water, none of it passed into her stomach, and all of it could be recovered through the tube which was in the sac. The diagnosis of right and left diverticulum of the esophagus being made November 25, the patient was advised to practise gavage, which she readily consented to do. On January 21, she again presented herself at my office, having improved rápidly since I last saw her. She had gained about 12 pounds in weight. The anemia had disappeared, as evidenced by the return of color to the lips, cheeks, and conjunctivas. She has had no pain at any time and feels strong and well. She has had no trouble from constipation during the past five weeks. On September 7, 1905, she again called at my office to report steady progress, with entire absence of all untoward symptoms. She has gained 14 or 15 more pounds, making a total of nearly 30 gained since she began tube feeding.

DISCUSSION.

DR. MORRIS MANGES, of New York City: In a case of diverticulum of the oesophagus that came under my observation, the obstruction had existed about fifteen years. Three years ago Dr. William T. Bull did a gastrotomy, and some time afterwards tried to pass an instrument from below upward through the cardia. It had been easy to pass an instrument down into the stomach prior to the operation, but on account of the resulting stenosis below the diverticulum it was utterly impossible to pass the instrument upward, although the man was able to swallow some food.

A CASE OF FATAL HEMORRHAGE FROM THE UPPER GASTRO-INTESTINAL TRACT, WITHOUT ANY GROSS ANATOMICAL LESION: WITH SPECIMEN. REPORTED BY DR. I. ADLER OF NEW YORK CITY.

The patient was a man, 66 years old, whose previous history was rather uneventful. He had been a very heavy smoker and a large eater, but with the exception of occasional rare and slight intestinal disturbance his heatlh had always been good. In the beginning of this year he took a vacation trip to the South and there became ill. The exact nature of the trouble could not be ascertained. Abdominal pain, vomiting, loss of appetite appeared to have been the chief complaints. He was treated for several weeks by local physicians, principally, as it would seem, with calomel and saline purgatives. On his return to New York he was taken in charge by his family physician, Dr. Waller, with whom I saw the patient a number of times and through whose courtesy I have been enabled to study the case more closely. When he arrived in this city the patient was very weak and anemic, had little or no appetite and complained of frequent diarrhea and vomiting and of an indefinite pain referred without strict localization to the region of the right hypochondrium. About a week after his return to the city tarry stools were noticed and soon also attacks of hematemesis. Examination of the blood showed a simple secondary but progressive anemia, accompanied at first by a very slight leucocytosis which, however, soon disappeared. The urine showed nothing essentially abnormal. Examination of the stomach contents after a test meal showed a slight hyperacidity, but otherwise perfectly normal conditions; no stagnation or retention, no disturbance of motility and no dilatation—in fact, the stomach was rather small. Fairly comfortable as a rule during the day, the man was usually disturbed at night by indefinite pains, by nausea and gas distension of the bowel. Sometimes every day, or at irregular intervals of one or more days, there would be bloody vomit, more or less profuse, either of rather fresh blood or, more rarely, of the typical coffee-ground. The stools were often tarry. The diagnosis of ulcer was made with a probable localization in the duodenum, and, in view of the man's age and history and his rapid failing in health and strength, with a suspicion of carcinoma. Medical treatment proved of no avail and as the bleeding continued and the patient rapidly declined, an exploratory laparotomy was decided upon and performed by Dr. Willy Meyer. The stomach was found to be very small and no lesion of any kind could be detected neither in the stomach itself nor in the duodenum. There were no signs of a malignant growth nor of enlarged glands. In view of the possibility of a recent simple ulcer in that part of the duodenum that

could not easily be explored, it was thought best to do a gastro-enterostomy, which was performed as rapidly as possible according to the McGraw method. The patient stood the operation very satisfactorily, showing hardly any effects of shock, and for the first twenty-four hours did exceedingly well. About thirty hours after the operation profuse hemorrhage occurred with repeated vomiting of blood and bloody stools, followed by rapid collapse and death. Permission for a complete autopsy could not be obtained, but the wound was re-opened and through this all the organs below the diaphragm were removed. Most careful scrutiny failed to detect any macroscopical cause for the hemorrhage. There were no signs of ulceration in the stomach; two small submucous hemorrhages in the neighborhood of the McGraw ligatures were probably the result of severe handling during the operation. They were well covered with epithelium and could not possibly have caused hemorrhage. There were no erosions, no varicose veins or aneurysmatic dilatation of arterioles. From the pylorus down to the anus no gross lesion of the intestines could be made out. There were no apparent lesions of the mesenteric or other vessels and the aorta was normal with the exception of very slight atheromatous patches. Liver and kidneys were apparently sound. The pancreas showed the slight fibrous induration corresponding to the age of the patient. The microscopic examination of the organs has not yet been completed and will be published later.

The case appears to be one of the very rare instances of hemorrhage from the gastro-intestinal tract without apparent lsion. Hemopholia, gastro-intestinal hemorrhage, ondary to systemic disease or to diseases of other organs as the liver, for instance, can, I think, be excluded. Though the autopsy was necessarily not complete, I believe that cardiac or pulmonary disease, or varix of the esophagus, may also, for reasons which I need not enter upon here, be excluded in this case. The few similar cases reported in the literature seem all to have occurred in subjects of advanced years. I am inclined to believe that the hemorrhages originated not from one single point, but probably from several, and perhaps both in the stomach and in the duodenum. I would like to seek the analogon of these cases in the hemorrhages, often so troublesome and not infrequently fatal, as they occur in the nose of senile and atheromatous subjects. In a profuse epistaxis it is often very difficult to find the bleeding points during life, and it seems to me absolutely impossible to find them in the corpse. The cause of these hemorrhages, therefore, is to my mind, most probably to be sought in the arterio-sclerotic condition of the small vessels, both arteries and veins, of the inner lining of the stomach and the upper intestine.

DISCUSSION.

DR. A. ROSE: I would like to ask Dr. Adler if there was any relaxation of the abdominal walls in the case he *reported?* My experience has convinced me that in any *case of haemorrhage* from the stomach or intestines, the

application of the plaster strapping will be of service. I recall a case of enteritis of at least eighteen months standing, in which the patient suffered very much from diarrhoea. Since the plaster strapping was applied she has been free from diarrhoea and can eat anything, even articles of food which before the strapping would have caused severe trouble, without a recurrence of her former symptoms. This shows unmistakably the effect of the strapping on innervation and circulation.

My observation, and that of others, has been that in cases of enteritis, with diarrhoea and haemorrhages, the plaster strapping will arrest the bleeding and cure the diarrhoea. Plaster strapping is something entirely different from the ordinary bandage support. In a case of dysmenorrhoea of ten years' standing, where the patient had been obliged to spend two days in bed every month during her menstrual epoch, the symptoms were entirely relieved by the plaster strapping.

In cases of haemorrhage, like that reported by Dr. Adler, it seems to me therefore probable that the plaster strapping suggests itself on account of its physiological effect on the circulaiton. The benefit from this method of treatment is not limited merely to the abdominal support; it can no more be compared to the ordinary abdominal bandage than the plaster-of-Paris bandage applied to a fracture or arthritis can be compared to an ordinary splint. The plaster strapping not only supports the abdominal viscera, but also calms the nerves and exerts a physiological effect on the circulation.

DR. T. B. FUTCHER, of Baltimore: To a certain extent, the case reported by Dr. Adler corresponds with the history one gets in cases of haemophilia, although the absence of a history of bleeding earlier in life rather excludes that diagnosis. The blood might not come from any single point, but from the gastric mucosa or from the oesophagus. It would have been interesting in a case of this kind to have known the coagulation time of the blood.

DR. MANGES: It is rather hazardous to draw any conclusions in this case, in view of the fact that the thoracic organs were not examined. In a case I saw some years ago, the patient died from a haemorrhage of this kind: there was no reason to suspect an oesophageal varix, and yet at autopsy a small eroded area was found in the oesophagus.

DR. ADLER: I saw no reason in this case to suspect that the recurrent haemorrhages came from an oesophageal varix, or other lesion referable to that organ. In the course of the operation, the finger was inserted into the cardia, and the lower end of the oesophagus appeared to be in normal condition. Furthermore, the stomach tube was passed repeatedly, and did not give rise to haemorrhage. The clinical history, the hyperacidity, the pain in right hypochondrium, etc., etc., all point to a lesion lower down.

As far as haemophilia is concened, there was no family or previous history to indicate that condition, nor any

lesions of the skin or elsewhere to lead one to suspect it. In the course of the operation it was noticed that there were an excessively large number of bleeding points in the muscles and subcutaneous tissue, but the blood clotted very nicely. We neglected to ascertain the coagulation time of the blood, but I doubt if it would have given us much help. There were no symptoms pointing to the thoracic organs.

I have had no experience in strapping the abdomen, to which Dr. Rose referred, and can offer no opinion as to its effect in cases of this kind. I can imagine that in cases where the abdominal organs are engorged with blood, owing to some disturbance of the splanchnic circulation, this strapping method, by compression, might exert some beneficial influence.

DR. S. J. MELTZER: In this discussion reference has been made to the clotting time of the blood in haemophilics. It is well to remind of the fact stated by Sahli that during the haemorrhage the coagulation time might be even shorter than in normal, but during the interval the coagulation time is prolonged in haemophilia. The fact that during a haemorrhage the clotting time is not prolonged does therefore not speak against the diagnosis that the bleeding might be due to haemophilia.

[Reprinted from *American Medicine*, Vol. IX, No. 22, pages 893-895, June 3, 1905.]

FURTHER REMARKS ON ISCHOCHYMIA AND ITS TREATMENT.[1]

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Ischochymia or "clinical dilation of the stomach" of the older authors is an important disease, to the recognition and treatment of which modern stomach pathology has made many contributions. In two previous papers[2] I have already touched upon the symptomatology of ischochymia of benign, as well as of malignant origin, and in a general way sketched its treatment. In the present paper, I would like first, to discuss the statistics of this affection, and second, to describe a few new points in the clinical course of ischochymia, as well as its treatment.

1. *Statistics.*—As to the frequency of ischochymia, I looked over my journal of 1904 and found the following figures: Total number of patients 3,243, of whom 1,793 were men and 1,450 women. Among these there were found 47 cases of ischochymia of benign or malignant nature, which were distributed as follows:

TABLE OF CASES OF ISCHOCHYMIA.

Variety.	Total number.	Men.	Women.
Benign	23	17	6
Malignant	21	13	8
Undetermined	3	2	1
Total	47	32	15

[1] Read before the American Gastroenterological Association, April 24, 1905, New York.

[2] Max Einhorn: Diagnosis and Treatment of Stenosis of the Pylorus. Medical Record, January 19, 1895. A Further Contribution to Our Knowledge of Ischochymia. Medical Record, June 19, 1897.

Estimated in percentages we get the following figures:

ISCHOCHYMIA OF BENIGN AND MALIGNANT NATURE.

Total number.	Men.	Women.
1.45%	1%	0.45%

ISCHOCHYMIA OF BENIGN NATURE.

Total number.	Men.	Women.
0.71%	0.52%	0.19%

ISCHOCHYMIA OF MALIGNANT NATURE.

Total number.	Men.	Women.
0.65%	0.41%	0.25%

The figures given would in reality probably be somewhat higher. As is customary, I examined the stomach by means of a tube only, whenever there was a suspicion of some graver lesion. Under these conditions it is naturally unavoidable that some cases of benign ischochymia should remain unrecognized.

2. *Benign Ischochymia, Associated with Severe Peristaltic Restlessness of the Stomach, Cured by Medical Means.*—It is generally well known that cases of benign ischochymia, which are caused by a moderate stenosis of the pylorus, may be improved or cured by medical treatment. In these cases the dilation of the stomach is not very great and the peristaltic restlessness of the organ is absent or only slightly present. There are, however, exceptions to these general rules, and occasionally cases of tremendous gastric dilation (filling the entire left side of the abdomen down to the symphysis) with marked peristaltic restlessness and ischochymia are cured by palliative treatment.

As examples we may cite the following two cases:

CASE I.—December, 1903. Harry H., aged 32, developed pleuropneumonia in September, 1903. Since then he has been suffering from severe stomach trouble. He had intense pains in the epigastrium, accompanied by nausea and vomiting as well as constipation. He lost in weight rapidly and his troubles became worse; the vomiting occurred several times daily and he brought up large quantities of fluid (over a quart of gastric juice mixed with food). When I saw him for the first time in December, 1903, he was very much emaciated. His stomach was much dilated, reaching down to the symphysis, and showed great peristaltic restlessness. In the left chest was still found the evidence of the inflammatory process of the lung (rales and dulness).

Besides the process in the lung, ulcer of the stomach with stenosis of the pylorus was diagnosticated. As the patient was much run down, medical treatment alone could be considered.

He was therefore placed upon a strictly fluid diet and

received internally magnesia usta, 10 gm.; bismuth subnitrate, 20 gm.; water sufficient to make 200 gm.; a tablespoonful three times daily a half hour before meals.

The patient then entered the German Hospital and was under my care from January 14 to March 30, 1904. I mention here the following extracts from the hospital records:

Diet: Milk or oatmeal with milk, 8 oz. every 2 hours; 3 raw eggs daily. Medication as above. Oil enemas. Heroin. Every other day lavage of the stomach in the fasting condition.

Lungs: In front on the left side above, some rales; on the left, behind in the middle of the scapula and the lobe, rales and dulness.

January 15, 1904. Aspiration under the left scapula produces a somewhat purulent fluid, containing microscopically many white blood cells, but no bacilli.

January 18. Thiocol 0.3 gm. three times daily. January 21. Patient was up for a half hour; weight, 102 pounds.

The diet is somewhat increased, two teaspoonfuls of farina being added to eight ounces of milk.

January 23, 12 ounces of milk every two hours; six eggs. February 5, diet as before, seven eggs; weight 104 pounds. February 9, respiratory exercises with James' bottle. February 11, weight, 104 pounds. February 15, diet as before; eight eggs. February 22, test-meal of rice in the evening; three soda crackers, raw oysters, and lamb chops. February 23, lavage of stomach in the fasting condition shows the presence of rice kernels. February 29, diet again increased by the addition of mashed potatoes, toast, butter, and two soft-boiled eggs.

Weight, February 25, 108¾ pounds. March 3, 113 pounds. March 17, 124¾ pounds. March 24, 127¼ pounds.

After the patient had left the hospital he continued to gain, and weighed 153 pounds in November, 1904. He was able to eat everything, and looked splendid. Examination revealed that the stomach did not reach below the navel.

Case II.—April, 1904. Mrs. G., aged about 30, had been complaining for the last five to six years of severe attacks of gastralgia and vomiting, which usually occurred periodically about every five to six months, and lasted from four to five weeks. The patient consulted me for the first time about three years ago. She was, at that time, much emaciated, and suffered intensely every time she took food. Examination revealed normal conditions in the thorax. In the abdomen, however, the stomach was found to extend from the left costal margin to the symphysis, as determined by the presence of capotement. Slight tenderness on pressure was found over the entire stomach, but no circumscribed area of localized pain. On inspection, prominences could be plainly perceived, traveling in an undulatory manner from left to right. Peristaltic restlessness of the stomach was evidently present. Examination of the stomach contents one morning when she had had only milk revealed a chyme that was very much decomposed, and had an odor of H_2S. It contained muscle fiber, pieces of cellulose and bread particles. Chemically, the stomach contents showed the presence of free HCl, increased acidity and hydrogen sulfid. The patient was placed on a strictly liquid diet and received magnesia usta, and bismuth.

Gradually her condition improved, and she began to gain in weight. During the first six weeks of treatment after taking a test-supper of rice and milk the rice was found on washing the stomach in the fasting condition the following morning. Later

the amount of residue became less and less. She could now eat mushy food and some white bread.

In the course of the following year she gained about 40 pounds. Later she became pregnant, had a healthy child, and remained well for two full years.

In 1903 the patient made a visit to Russia. On board of the ship she ate corned beef and other heavy food. She again began to suffer from gastralgia and vomiting. She was treated in a hospital in Warsaw without her condition improving.

In the fall of 1903 she returned to New York and came to my office. She was again very much emaciated, weighed 90 pounds, had severe pain, and vomited daily large quantities of foul-smelling food.

Examination revealed a condition similar to the one of 1901: Stomach reaching down to the symphysis, showing strong peristaltic movement. The contents had the odor of H_2S, showed the presence of food from the previous day, the presence of HCl, and an increased acidity. Under the same treatment as that of 1901, the condition of the patient improved gradually. In the course of nine to ten months, the patient has gained over 50 lbs. At present the stomach, washed out in the morning in the fasting condition after a test-supper of rice, is always empty. The patient can readily digest all ordinary articles of diet. She must therefore be looked upon as cured.

The cessation of the symptoms due to the stenosis may best be explained as follows: Let us assume that the pylorus is only slightly narrowed (relative stenosis of the pylorus), so that the usual food passes readily. Through some grave dietetic error an inflammation of the pyloric part takes place, which causes a hyperemia, perhaps an edematous swelling of this part of the stomach, and the result is that the canal is nearly impassable. The whole process may best be compared to an angina of the throat. If now, by means of rest and suitable treatment, the acute swelling is made to disappear, the former lumen of the canal will be reestablished.

3. *Cases of Benign Ischochymia with a History of Short Duration.*—Usually in cases of benign ischochymia the patients give a long history of suffering, with periods of euphoria interspersed, whereas cases of malignant ischochymia show a short history of illness (three to eight months) with constant suffering. There are, however, exceptions to this rule, and I would like to describe two cases of benign ischochymia with a relatively short period of illness:

CASE I.—December 24, 1902. Rev. Ch. McK., aged 63, complained for the last three to four months of digestive disturbances. He has pains in the upper epigastric region one to two hours after meals, vomits large quantities of food at times, is constipated, and is steadily losing weight. In all, he has lost 25 pounds in the last three months.

Examination reveals nothing abnormal in the thorax. In the abdomen we find a markedly dilated stomach with a doubtful resistance; the contour of the colon, which is tender on pressure, can also be made out. Examination with the stomach-tube shows the presence of food remnants from previous days, presence of free HCl, and an acidity of 72.

For two weeks remnants of food from the previous day could be detected on emptying the stomach in the morning in the fasting condition.

On liquid diet the condition of the patient gradually improved. Soon he was able to consume semisolid, and later solid food, and felt perfectly well. Later he regained his original weight and has been well since.

CASE II.—April 3, 1904. Joseph P., aged 32, wood-carver, has always been well. Two years ago he complained for a short while of vertigo, belching, and loss of weight. He was better again, and felt well up to half a year ago, when he again began to complain of vertigo, belching, and loss of weight. He lost over 30 pounds in the last few months. Appetite and bowels are good. He has no pain nor does he vomit, but feels so weak he can hardly walk.

Present state: Patient looks pale. Nothing abnormal can be detected in the thoracic organs. In the abdomen there is no sign of a tumor nor any localized tenderness. The stomach reaches two fingers' width below the navel. Examination with the stomach-tube shows food remnants from the day previous. Free HCl is present, blood and lactic acid are absent.

The patient was placed upon a liquid diet, and the stomach washed in the fasting condition every other day. During the first week of treatment, food remnants were constantly found, later the stomach was always empty in the fasting condition.

He was then told to eat semisolid, and later solid food. He recovered completely.

4. *Cases Appearing Clinically as Malignant, at times Reveal Themselves as Benign.*—We have just referred to a category of cases of ischochymia which, in spite of a history of short duration, usually pointing to malignancy, are of a benign nature. There is one more characteristic, making the diagnosis of malignancy a positive one, and that is the occurrence of a distinct tumor in the stomach or pylorus. Considerable gastric disturbances of a few months' duration, associated with ischochymia and the presence of a tumor, are universally considered as diagnostic of cancer of the stomach. I have, however, observed in my practice two cases in which we had to deal with these conditions, and in which the tumors were seen and palpated during operations and were clinically recognized as certainly cancerous, which, according to their future course were certainly not malignant. These two cases I shall describe:

CASE I.—October 24, 1896. Samuel K., aged 36, has always been well until six months ago, when he began to suffer from stomach trouble. He had pains almost after every meal, used to belch a great deal, and later vomited. Obstinate constipa-

tion was present. He continued to lose steadily in weight (in all 40 pounds) and strength.

Examination showed a markedly dilated stomach, with visible peristaltic restlessness of this organ. The stomach-tube showed the presence of food from the day before; HCl was present, but no lactic acid.

In view of the presence of ischochymia, the peristaltic restlessness of the stomach, and the relatively short duration (half a year), the diagnosis of pyloric obstruction due to cancer was made, and an operation was advised. He was operated upon by Dr. Willy Meyer, on November 6, 1896, at the German Hospital. After exposing the stomach, a tumor involving the pylorus and extending toward the duodenum, was found. There were also small whitish infiltrations of the size of a pea in various places in the upper part of the jejunum. It did not seem advisable to resect the entire portion of the stomach and bowel involved, and gastroenterostomy was therefore performed. The diagnosis made during the operation was cancer of the pylorus and secondary deposits in the bowel. The patient, however, got well, and is still (eight to nine years later) in the best of health.

The tumor could, therefore, not have been a cancer. Perhaps we here had to deal with a tuberculous neoplasm and diffuse tubercles in the bowel, which probably disappeared after the laparotomy.

CASE II.—August, 1901. Thomas G., aged 45, was always well until six months ago, when he began to suffer from dyspepsia. He lost his appetite, felt heavy after meals, and occasionally had severe pains in the epigastrium. Later, constipation and vomiting appeared. He lost steadily in weight and became much weaker.

Examination revealed a tumor the size of an egg in the region of the pylorus, and a much dilated stomach, presenting signs of ischochymia. HCl and an increased amount of acidity were found in the stomach, as well as food remnants from the previous day. Sodium iodid was tried for two weeks, but without success. The diagnosis of cancer of the pylorus having been made, an operation was suggested and performed on September 10, 1901, by Dr. S. Lloyd, in my presence, at the New York Postgraduate Hospital. Inspection of the stomach revealed marked enlargement of this organ and a tumor, the size of a fist, at the pylorus, which was adherent to the surrounding organs. It seemed impossible to extirpate the tumor, and gastroenterostomy, by means of McGraw's elastic ligature, was done.

We all were of the opinion that the patient would soon succumb to the disease. This, however, did not happen. The patient got well, and has remained so up till now, having gained considerably in weight. As four years have elapsed since the operation, we must assume that the tumor, which clinically and macroscopically showed all evidences of cancer, was not a cancer, but something else.

5. *Cases of Benign Ischochymia, with Ulcer at the Pylorus and Continuous Hypersecretion.*—We occasionally encounter cases of benign ischochymia, in which the following three stages of the disease may be observed: At first there is for years hyperacidity, resembling the ordinary form, being, however, more difficult to treat

and showing a tendency to recur. We then notice that a condition of continuous hypersecretion of the stomach has taken place. The pains are now more intense, vomiting may be present or absent. After the stage of continuous hypersecretion has lasted some time (a year or more), a new factor, that of ischochymia, is added. This forms the third and gravest stage of the disease. In these cases we usually have an ulcer near the pylorus (either in the stomach or in the duodenum). It is often clinically observed that shortly previous to or after the appearance of the ischochymia, hemorrhages of the stomach occur.

These cases of benign ischochymia, with stenosis of the pylorus and ulceration, represent, according to my experience, a severe type of the disease, *i. e.*, medical treatment is usually of no avail, and an operation (gastroenterostomy) is usually indicated.

In the last few years I have observed six pronounced cases of this type. In all of them an operation was urgently advised, as the ischochymia continued even after rectal feeding and liquid diet.

Three of the patients gave their consent (Charles L., Otis P., and Samuel L.) and were operated upon. Of these, one (Charles L.) died five days after the operation with comatose symptoms, whereas the other two (Otis P. and Samuel L.) recovered and were entirely free from all symptoms. Three of the six patients (S. S., Theodore L., and Sigm. H.) were decidedly against any operative interference. Of these latter two died, S. S., from a perforation of the stomach and Theodore L. from a duodenal hemorrhage, the third (Sigm. H.) slowly improved and regained his health almost entirely. The history of the last case I would like to give in detail.

October, 1902.—Sigm. H., aged 36, merchant, had been suffering for five to six years from periodic attacks of severe hyperchlorhydria (pains two to three hours after meals; relief after partaking of food and drink). A course of treatment at Carlsbad brought him only partial relief. These attacks were worse at the time when he was overwhelmed with work. On examination a greatly dilated stomach was found which reached a hand's width below the navel. There was marked hyperacidity, but no disturbance of motor function. The stomach at that time was completely empty in the fasting condition. Patient was treated with the customary alkalies and improved.

In January, 1903, the patient reappeared. He said that for the last four to five weeks he had severe pains in the stomach and frequently woke up at night and after having considerable paroxysms of pain had to vomit. The ejected matter usually

consisted of a rather clear fluid, in which only rarely particles of food were found, and which was strongly acid.

Examination with the stomach-tube in the fasting condition produced about 100 cc. of pure gastric juice without any food particles. On February 3, 1903, food remnants were found for the first time in the fasting stomach, besides a larger quantity (150 cc.) of gastric juice. At the time there was also peristaltic restlessness of the stomach.

Patient was placed upon a strictly fluid diet and was treated with large doses of magnesia usta and subnitrate of bismuth. He felt somewhat better, and on February 22 the stomach washings in the morning were free from food remnants. There was always, however, a large quantity of pure gastric juice (100 cc. and more). Its acidity was not very high, oscillating between 60 and 80, the total acidity on March 30, 1903, being 60.

In the night of April 3 and 4, 1903, a severe gastric hemorrhage suddenly occurred. He vomited large quantities of blood and fainted in the toilet. Later bloody stools were passed and another hematemesis occurred. Patient was treated with adrenalin and nutritive enemas and his condition again improved.

The diagnosis of ulcer involving the pylorus was made, and in view of his long-standing trouble, an operation (gastroenterostomy) was advised. Patient did not, however, consent to this and was, therefore, treated further in a palliative manner, his condition improving visibly.

On April 29, 1903, the stomach was found empty in the fasting condition. The diet now was carefully increased and he was sent to the country. In October, 1903, I again saw the patient. He felt perfectly well, had gained 25 pounds, and was able to eat ordinary food without any difficulty. He has since remained well.

6. *Treatment.*—There are two ways of treating ischochymia: (1) Dietetic and medicinal measures (rectal alimentation, fluid diet, lavage of the stomach, bismuth, etc.); (2) operative procedures (gastroenterostomy, pyloroplasty).

These two methods of treatment do not antagonize but supplement one another. The indications for both are fairly well determined, where one ceases the other begins.

Since in by far the larger number of cases of ischochymia a stenosis of the pylorus is present, the ideal method of treatment would consist in forming a new passage for the exit of the chyme from the stomach. Yet surgical intervention ought not to be recommended immediately in every case, as a certain element of risk is still attached to this procedure. The mortality of gastroenterostomy and pyloroplasty is rather high. It varies among different surgeons and in different countries between 5% and 20%. If we assume 10% as the average (among my own patients the mortality was much higher), we see that we have a mortality percent-

age that ought to be taken into consideration in advising an operation.

The indications for medical and surgical treatment of ischochymia may be placed as follows:

1. Benign ischochymia requires first medical treatment; if this be unsuccessful, *i. e.*, if after a longer period of treatment, the fasting stomach, on a fluid diet, is not empty, but contains food remnants, an operation is advisable.

2. Surgical intervention is also indicated in benign ischochymia which has developed subsequent to a condition of continuous hypersecretion of gastric juice (preceded by hemorrhage or not).

3. Malignant ischochymia or one of dubious nature in which, however, a thickening of the pylorus is found, should also be treated surgically (gastroenterostomy, and if possible, resection of the pylorus).

The reasons for the first two postulates are clear from the preceding article. Benign ischochymia should first be treated by dietetic and medicinal measures, because many patients with apparently grave cases of this kind frequently get well in this way; and second, because an operation is a procedure connected with a considerable amount of danger, and should be suggested only when absolutely necessary.

Those cases of ischochymia with preceding gastrosuccorrhea form an exception, and require operation sooner, because they are generally complicated with active ulcerated processes in the pyloric region, and because they are frequently accompanied by severe complications (perforations and severe hemorrhages). The danger from operation in this variety of ischochymia is less than that from possible complications, therefore an operation is indicated.

As regards the third class of cases, referring to malignant ischochymia, operative procedures must be recommended, first, because, these cases grow progressively worse, and second, because a possibility of a radical cure (either by extirpation of the tumor or in consequence of the disappearance of the same after gastroenterostomy), even if remote, is given.

At the close of this article I would again like to emphasize that both the medical and dietetic treatment as well as the surgical treatment, are of great value in overcoming ischochymia; they are not antagonistic to each other, but work hand in hand—where one ceases the other commences.

DISCUSSION.

DR. A. ROSE: The name ischochymia, used by Dr. Einhorn in his paper, is misleading. Ischo means to check or arrest something by force, where the force is actively applied. The term, therefore, is not appropriate in a case where there is passive obstruction, like stenosis or atony. He has taken the word from ischuria, where the retention of the urine is caused by a spasm of the circular or longitudinal fibres, but the word ischuria would not be appropriate in a case where the retention of the urine was due to paralysis of the detrusor vesicae.

DR. J. KAUFMANN, of New York City: As this subject will come up for discussion to-morrow, I do not want to go into it to-day, but I only wish to confirm Dr. Einhorn's statement that we see certain cases of benign stenosis get well under medical treatment, especially by the use of lavage. I have seen such cases. However, this is nothing new. If Dr. Einhorn will give himself the pleasure of reading the first article by Kussmaul on the treatment of dilatation of the stomach, by the use of the stomach pump, he will find the description of such cases. It was the excellent results obtained by Kussmaul in this very class of cases that made lavage of the stomach so popular. Kussmaul reported cases of far advanced stenosis that recovered entirely under lavage.

DR. H. W. BETTMANN, of Cincinnati: I think it very important that the profession should know that even severe cases of ischochymia, with obstruction at the pylorus, do get well under medical treatment. The surgeons are too apt to overlook this fact, and to recommend surgical interference rather recklessly in some cases. It is of advantage to the profession at large, and of especial value to the patients to know, that these cases of obstruction with dilatation have a fair opportunity of getting well under medical treatment. My own experience has led me to persist for a long time with the medical treatment, rather than recommend early surgical interference. My method for many years has consisted in giving patients with obstructive dilatation two very large meals in the course of twenty-four hours, with an interval of about ten or fourteen hours between them, and washing out the stomach about half an hour before the meal is given.

According to my observation, the obstruction in these cases is (1) an actual anatomical narrowing made up of 3 distinct factors, all of which co-operate in nearly every case; (2) a spasmodic obstruction; (3) stagnation, due to muscular weakness. If the obstruction is an anatomical one, we cannot remove it by medical treatment, but we can relieve the obstruction due to the other two causes. We should order a diet that will not prove irritating, meat, milk, buttermilk, etc., and give it in a liberal quantity, even as much as two quarts. Twelve hours later this is repeated, preceded by lavage. In this way the general condition of the patient is often marvelously improved in a short time. By giving bland and concentrated foods, and by employing lavage, we relieve the spasmodic contraction

at the pylorus, which is usually due to the irritation of stagnating and decomposing foods. By giving sufficient nourishment we build up the patient's strength and, with it, the muscular power of the stomach. In this way we often find what we took to be an irremediable anatomical stenosis yield completely to medical treatment and the patient is thus restored to perfect health. It is not possible to tell at the outset in any given case how much of the stenosis is due to anatomic changes and how much is merely functional. It is therefore advisable to recommend a rational medical treatment before turning the patient over to the surgeon.

DR. EINHORN: I read Dr. Kussmaul's article, to which Dr. Kaufman referred, long ago, and have frequently mentioned it in writing on this subject. I know very well that dilatation of the stomach, with obstruction, can be cured, and in this paper I mention the fact that even the gravest cases, in which at first sight surgical interference seems imperative, may get well under medical treatment.

In reply to Dr. Bettmann I would like to say that cases of ischchymia, due to muscular weakness, are extremely rare. In about 95 per cent of cases we have to deal with a real obstruction. In the cases that I had operated an actual obstruction was always found, and I believe that even in the earlier stages of the disease we have a real anatomical lesion (relative stenosis). The stenosed portion at times may become inflamed and close up the opening entirely, and then we get these abrupt, severe symptoms. Under rest and proper dietetic treatment the inflammation subsides and the patient again gets along fairly well.

Some years ago it was my usual custom to have these patients operated on, but the mortality was so high that I became more cautious.

With regard to the correctness of the term ischochymia, Dr. Rose may be right in his contention. I took it from the word ischuria, to which condition Kussmaul compared it. The stomach is overloaded with chyme, and that is the main point.

[Reprinted from THE MEDICAL NEWS, July 29, 1905.]

PRIMARY SARCOMA OF THE STOMACH.[1]

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ALTHOUGH it is true that sarcomata must be included among the rare tumors of the stomach, yet recent studies have demonstrated that they may possibly occur more frequently than is generally supposed. Fenwick is probably not far from the truth in his assertion that they constitute from 5 per cent. to 8 per cent. of all gastric tumors. In proof of his claim he urges that the greater number of cases has been reported since attention was especially called to their occurrence by Virchow, forty years ago, and that the re-examination of some specimens which had been catalogued as gastric cancer has demonstrated that the tumors were really sarcomata. Furthermore, that although Schlesinger was able to collect only 30 cases in 1897, yet in November, 1900, the number of cases exceeded 60. Rosenheim also states that sarcomata are not as rare as is supposed. Perry and Shaw found 4 cases of sarcoma in 50 cases of malignant disease of the stomach; Haberkahndt found 3 sarcomata in 207 pyloric resections; while Czerny found only 2 sarcomata in 298 gastro-enterostomies.

The two cases which I shall report occurred within one year at the Mt. Sinai Hospital. Dr. Harlow Brooks informs me that in addition to the case which he published in 1898 he has since been able to collect 3 additional cases during nine years' service at Bellevue Hospital.

That uncollected cases may be scattered through the literature was strikingly shown to me recently when, while hunting up a false reference

[1] Read at the Eighth Annual Meeting of the American Gastro-Enterological Association, held at New York, April 24 and 25, 1905.

in the transactions of the London Pathological Society, 1889, I found an unreported case of diffuse gastric sarcoma.

On the other hand, Alesandri, whose article is one of the most recent on the subject, still maintains that they are very rare. This question is of interest, not so much from a diagnostic and pathological standpoint, as to that of prognosis, since the results of operation in some cases of sarcoma are decidedly better than in carcinoma as is well shown in Dock's case in which a large lymphosarcoma was removed over four years ago. I have learned from a recent personal communication from Prof. Dock that the patient is still alive and well. It is possible, therefore, that in the absence of more definite pathological details on the subject, some of our observations on the operative results of carcinoma of the stomach may be modified.

To consider many of the details of the pathology, symptomatology, etc., is scarcely necessary, since much of it would be a useless repetition of what has already been well presented by Schlesinger, Fenwick, Alesandri and other writers. Here it will suffice to call particular attention to certain practical features:

1. That the new growth arises from the submucosa or muscularis, the mucous membrane being involved only late in the disease as a secondary result, or it may escape entirely. The curvatures, especially the greater, are usually involved, the pylorus being less frequently attacked.

2. The growth may be diffuse, as usually occurs in lymphosarcoma, or it may be circumscribed, this being the ordinary form of the spindle-celled type. These circumscribed growths often assume a polypoid form, growing either into the lumen of the stomach, or outwardly into the greater or lesser peritoneal cavity. These polypoid tumors constitute a very puzzling group, the diagnosis of which is exceedingly difficult, since it is in these cases that the mucous membrane is usually intact while few if any of the symptoms draw attention to the stomach as being the real source of the neoplasm.

3. The orifices are rarely stenosed and if the pylorus is involved the stenosis never reaches the degree so frequently encountered in carcinoma.

4. Degenerations frequently occur in these tumors. These include hemorrhages, cystic degeneration, formations of fistulous tracts, etc. On the other hand, it is to be noted that sometimes the sarcomata are themselves degenerations of other benign tumors such as fibromata, myomata, etc.

5. As regards size, they may constitute large tumors (weighing 12 pounds or more), or they may be so small as to be found only by mere chance at autopsy.

6. They may be primary or secondary. The latter are the rare forms, except in lymphosarcoma where the secondary forms are commoner than the primary. Doubt has even been cast upon the cases of primary lymphosarcoma since it is urged that some small, primary lymph-gland tumor may have escaped notice. These secondary lymphosarcomata are usually derived from some growth in the neck, pharynx, gums, rectum, retroperitoneal sarcoma, etc., or they may constitute part of a general lymphosarcomatosis of the gastro-intestinal tract, or they may be an internal manifestation of Hodgkin's disease.

7. Metastases may occur in neighboring or distant organs, or they may be absent. The distant organs may include the skin, testicles, tongue, tonsils, rectum, etc. The skin metastases are of particular interest since their occurrence is much more frequent than in cancer and may thus afford much important diagnostic information.

8. *Age.*—The range is very great. Finlayson's case occurred at 3½ years and the case of Malvoz occurred at seventy-eight. Fleiner states that they may even be congenital. As a rule the average age is younger than in carcinoma; thus the 4 cases of lypmhosarcoma reported by Perry and Shaw were fifteen, eighteen, thirty-eight and sixty-seven years old.

Special attention may be directed to a group of cases with gastric symptoms in which hematemesis is the leading symptom. Although it has been shown that bleeding is a relatively infre-

quent symptom in gastric sarcoma and is used as one of the points of differential diagnosis, yet cases have been reported in which the bleeding was either an early or a late symptom and even led to that mistaken diagnosis of gastric ulcer.

One of the earliest cases with hematemesis is that of Hartley to whom I am indebted for the following report:[1]

Mrs. B., fifty-four years. Five years ago (1890), without any special cause, she vomited a large amount of coffee-ground material. The vomiting did not recur and she went on for four years in fair health, except for the heavy feeling after eating. One year ago she again vomited blood of coffee grounds and clotted in character for five mornings in succession; this was again without cause or exacerbation of gastric symptoms. The pain in the back which she had suffered previously became much worse at this time and she noticed a considerable swelling of the stomach.

Physical Examination.—In the left lumbar and left umbilical region is a rounded tumor about the size of the adult fist, which is hard and elastic, freely movable and easily displaced up into the left hypochrondriac region.

Operation.—April 26, 1895. Incision 5 in. long, just to the left of the left rectus; the hand was introduced and prolonged search made. The kidney could be felt in place, the large intestine bulged into the wound and was normal; behind the mesocolon and in what appeared to be the position of the spleen was the large tumor; the spleen was not felt. The mesocolon was torn through and the surface of a tumor in lesser peritoneal sac disclosed. This tumor was brought through this opening in the mesocolon and it was seen that it arose from the posterior wall of the stomach by a pedicle 1½ in. in diameter. The stomach was pulled well out and graped by house surgeon well below the pedicle. The stomach was now cut through entirely around the pedicle and a circular part of stomach wall excised. Abdominal wound closed in usual manner with drainage in center.

1 This is the case which was published in the Annals of Surgery, 1896, Vol. 23, p. 609.

Pathological Report.—By Dr. Ferguson, May 10, 1895. The material is a pear-shaped tumor measuring 15 x 10 x 8 cm. Its surface is smooth but nodular. It is apparently covered by peritoneum. The tumor is cystic, the cysts varying in size from ½ to 4 cm. in diameter. The tumor is firm in consistency and of pale gray color. An area of stomach wall 5 x 4 cm. has been removed with the tumor, the mucosa has not been ulcerated but is intensely hyperemic. Sections show the ordinary appearance of spindle-celled sarcoma. The vascular supply is abundant, some of the vessels being large with thin walls.

Discharged cured, June 1, 1895.

Case II, which I present, was also marked at its close by severe hemorrhages from the stomach and bowels.

Oberst has just published a paper in which he directs especial attention to this group of cases; he reports a case of gastric sarcoma (man, twenty-five years old) which was mistaken for a gastric ulcer. He also quotes a similar case of Robert and two of Kundrat. He therefore urges that the possibility of gastric sarcoma should be considered in youthful individuals with hematemesis in which the course is unusual or in which there is delayed convalescence after hemorrhage of the stomach.

Symptoms.—The cases may be divided into two great groups: First, those without; and second, those with gastric symptoms.

In the first group are included the cases in which the tumor grows outwardly into the peritoneal cavity and also many of the cases in which the growth is toward the gastric lumen but in which the infiltration of the stomach does not involve the pylorus and leaves the mucous membrane intact. This group includes the unusual cases of abdominal tumors which have been incorrectly diagnosticated, the true condition being revealed at operation or at autopsy.

The complications from the peculiarity of the growth may dominate the picture, such as adhesions, torsions, displacements, hemorrhage in the tumor, cystic degeneration, suppuration, etc., or, metastases may cause leading symptoms such

as disturbances in the intestines, rectum, etc. One peculiarity of the intestinal metastases is to be noted—that they rarely cause stenosis, being thus unlike the secondary growths which occur in gastric carcinoma in which stenoses may much more frequently occur.

In the second group the gastric symptoms are practically those of gastric cancer. In most cases, however, the gastric symptoms are mild and only become marked toward the end of the patient's life.

A few points of differentiation may be noted. That vomiting and hemorrhage are less frequent and are usually only late symptoms. On the other hand, pain, fever, albuminuria, marked anemia and weakness are more marked and occur much earlier; indeed, the marked anemia and weakness may for a long time be the only symptoms. The appetite may fail, or, as in gastric cancer, may persist throughout. Perforation occurs more frequently. Marked splenic enlargement is much more frequently noted than in gastric cancer. On the other hand, omental and peritoneal involvement are less common. The skin metastases have already been referred to. A number of other minor conditions have been reported, still they are too inconstant to be of great differential diagnostic value.

The physical signs are inconstant as is readily understood from what has already been said concerning the pathology. Fenwick states that tumor was present in only 30 per cent. of the cases, and when the tumor was present it was frequently mistaken for tumor of the spleen, of the pancreas, kidney, omentum, ovary or retroperitoneal glands.

There are great variations in the size of the stomach since it may be enlarged or contracted or displaced or distorted, as occurs in the unusual cases where there is marked downward displacement of the stomach which is at the same time distorted into a sausage-shape resembling the transverse colon.

The stomach contents have been observed in about ten cases. They are practically identical with those found in cancer of the stomach. In

one of the cases which I report the same is to be noted. In one case which was under observation Erlach could follow the transition from normal chemism to complete absence of hydrochloric acid which occurred later in the disease. In several cases there was no change in the gastric secretion. Indeed, considering how long the mucous membrane remains uninvolved it is surprising that there is as much change in the chemism of the stomach as has been reported.

The stagnation in the stomach contents is not due so much to pyloric obstruction in these cases as it is to the infiltration of the muscularis.

Diagnosis.—The possibilities of diagnosis are best summed up in the words of Leube who has allowed them to remain unchanged even in the recently published edition of the *Diagnostik:* "In my opinion these tumors *cannot be diagnosticated,* not even when the conditions are very favorable for making a clever diagnosis, because of of the presence of a general sarcomatosis or cutaneous sarcomata. In one such case, it is true, I found a gastric sarcoma, but in another case of skin sarcomatosis the tumor in the stomach was a true epithelial cancer." However, if more exact points of differentiation are desired, I may quote the differentiating points given by Fenwick, who claims that "a diagnosis of round-celled sarcoma of the stomach may often be made by attention to the following points:

1. The disease usually occurs before thirty-five years of age, so that the younger the patient the greater the probability that the malignant affection is sarcomatous in character.

2. In many cases there is slight but continuous pyrexia, accompanied by rapid and profound anemia, while in carcinoma fever is usually absent during the early stages of the complaint and the cachexia much more gradual in its development.

3. Enlargement of the spleen is by no means infrequent but is rarely met with in cancer unless the organ is involved in the growth.

4. According to Kundrat the tonsils are apt to enlarge and the follicles upon the sides of the tongue may become swollen or ulcerated.

5. Secondary deposits in the skin occur in a notable proportion of the cases and permit of excision and microscopic examination. It should be remembered, however, that sarcomatosis has been met with in true cancer of the stomach (Leube).

6. A large nodular tumor due to infiltration of the omentum and a greatly enlarged liver with secondary growths in its substance are rarely met with.

7. Persistent albuminuria is often observed in sarcoma but is exceptional in cancer.

8. The discovery of pieces of morbid growth in the vomit renders the diagnosis certain. (Riegel, Westphalen).[1]

Prognosis.—As already stated, this in some cases is decidedly better than a carcinoma; in other cases, however, the course may even be more malignant.

Treatment.—If the diagnosis has been made arsenic ought to be used as it is in all cases of lymphosarcomatosis, otherwise the treatment is strictly surgical.

The histories of the two cases I present are as follows:

Case I.—L. S., shoemaker, forty-eight years of age, was admitted to Mt. Sinai Hospital to the service of Dr. Alfred Meyer, September 26, 1903. His symptoms are of four months' duration—loss of appetite, occasional vomiting of food, obstinate constipation. He has lost considerable flesh and strength. General condition fairly good; tongue moist, coated; a few small glands in the neck, axilla and groin. Physical examination negative throughout. Abdomen lax, tympanitic, no tenderness, no tumor palpable; general visible peristalsis. No fever. Urine negative. September 27—Test breakfast (Ewald).—Slightly digested; total acidity 40 per cent.; free hydrochloric acid absent; combined acids 30 per cent.; no lactic acid. Ferments absent.

September 30—Total acidity 84 per cent.; free hydrochloric acid absent; combined acids 26 per

1 Schlesinger made the diagnosis in one of the cases from a piece excised from a metastatic mass in the rectum.

cent.; lactic acid present; no blood. Ferments absent.

He left the hospital October 2 very much improved. He was readmitted on December 7. For ten weeks previous to his readmission he had had fever, pain in the upper right quadrant of the abdomen, the pain having no relation to the ingestion of food. Eructation and cardiac palpitation after eating. Progressive loss of flesh and strength. Vomited only once; no blood. Defecation and urination normal. Chief complaint abdominal pain and weakness.

Physical Examination.—Mucous membranes pale; pupils unequal, the left being somewhat larger than the right. Abdomen: The veins on both sides of the lower abdomen dilated. Liver extends to three fingers below the free border In the right hypochondriac region a large mass, somewhat irregular in shape, with a sharp edge can be felt; it extends on the right to the mammillary line, on the left it reaches one-half inch to the left of the middle line; its lower border is within one-half inch of the umbilicus; it is dull tympanitic on percussion and not tender; otherwise the abdomen is negative. Urine negative.

Blood Examination.—Thirty per cent. hemoglobin; red blood cells, 4,000,000; white, 13,600.

Test Breakfast (Ewald).Two ounces removed; fairly digested; total acidity 26 per cent.; free hydrochloric acid absent; lactic acid present; ferment absent.

December 10—The patient vomited a very large amount of undigested food. The mass became much more prominent after this and extended from the right mammillary line to the left parasternal line, above it reached the free border of the ribs, on the right of the mammillary line. midway between the umbilicus and the xiphoid was a sharp edge. Dull tympany over the tumor.

December 12—Movable dulness in both flanks, fluid in the abdomen. The patient is now running a slight evening temperature. The urine now contains a faint trace of albumin. For the past few days he has had three to four stools daily.

Consent having been obtained for an operation he was transferred to Dr. Gerster's service where

a gastro-enterostomy was performed on December 14. Death three days later.

Autopsy seventeen hours after death by Dr. Libman.

Murphy button found at site of gastro-enterostomy, good apposition of stomach wall and jejunum.

Lungs.—Right upper lobe was in incomplete gray hepatization. Both lungs adherent; markedly anthracotic. Pleura showed a moderate number of hard thickenings, some flat, some nodular. Acute pulmonary edema.

Stomach.—Moderately dilated. At pylorus a large, hard nodular tumor adherent to pancreas and duodenum and involving the whole circumference of the stomach for a distance of 3 inches from pylorus. Parts of the tumor were soft and had undergone degeneration. Pylorus admitted one finger. The interior of the stomach showed the same tumor ulcerated and involving the pylorus and stomach but not the duodenum. In other areas of the stomach wall there were some small hemorrhages in the mucosa.

Liver.—Normal size and consistency. Cloudy swelling (no metastases).

Kidneys.—Capsule of the right adherent. Both acutely degenerated.

Microscopic examination of the gastric tumor proved it to be a lymphosarcoma.

Case II.—Mrs. A. S., forty-eight years of age admitted to Mt. Sinai Hospital, under the care of Dr. Albert Kohn, August 6, 1904. For years this patient had had repeated uterine hemorrhages supposedly due to fibroids, for which, however she had refused operation. Not long before she came under treatment she had several uterine hemorrhages and her intense anemia was supposed to be due to that cause.

She was first seen July 3 with the symptoms of a marked secondary anemia, confirmed by several blood examinations. Loss of flesh and strength were not marked. Complaints were of extreme dizziness, sinking sensations, inability to assume the upright position. Headaches general. Loss of appetite, nausea, eructations. Vomiting only once or twice. She had many movements (9 to

12 daily) which were very loose, tarry and controlled with difficulty. Tenderness over the entire abdomen with pain, both marked over an epigastric tumor which was the size of a peach. The tumor was freely movable and did not disappear under the ribs. Her temperature was never above 101.5° F.; never subnormal.

Blood Examination.—Hemoglobin 18 per cent.; red cells 1,590,000. A few poikilocytes and microcytes; granular degeneration of red cells; moderate relative leucocytosis. Stools contain pus and red blood-cells. Hemin test positive. Urine 1.010, large trace of albumin; no tubercle bacilli.

The diarrhea, loss of appetite, nausea, eructation and obstinate loose, watery stools with small blackish fecal particles, the increasing anemia and abdominal pain and tenderness were the only symptoms. During observation at the hospital the most prominent symptoms were sighing respiration and weakness, diarrhea and vomiting, melena and bloody vomit. The physical examination showed not only a mass in the epigastrium, but two masses, one in each iliac fossa which were apparently new growths. However, on post-mortem examination nothing could be found to account for these two tumors. Test-meals could not be given on account of the wretched condition of the patient. She died August 13, 1904.

Autopsy.—By Dr. Buerger. *Lungs.*—Congested at base; few pleural adhesions. *Liver.*—Grayish-brown color, pale, firm consistency; marked parenchymatous degeneration, pigmentation, slightly fatty. *Stomach.*—Externally negative except for a somewhat depressed, scar-like area, 4 cm. proximal to pylorus, in the immediate neighborhood of which are stellate dilated vessels. Capacity somewhat increased; wall slightly thinned. About 4 cm. from the pylorus on the anterior wall there is a soft grayish-pink flattened sessile tumor, almost circular in outline, raised by a rounded though irregularly lobulated convex surface 12 mm. above the surounding mucosa. It appears even less prominent than this owing to a very marked, raised, somewhat cir-

cular, well-defined margin of slightly cicatrized mucosa immediately surrounding it. At one portion it is ulcerated and excavated so as to be on a deeper level than the mucosa of the stomach. Upon section it presents a radiate, irregular, streaky surface, here and there beset by small hemorrhages.

Microscopic examination shows it to be a myosarcoma. Intestines practically negative except for the presence of altered blood. In the ileum there are several script-like areas of brown pigmentation. No ulcerated areas or any points of bleeding could be found. Uterus contains small submucous fibromata. Bone marrow red in color, soft, diminution of fatty elements. Kidneys, intense parenchymatous degeneration. Interstitial changes in medulla. Spleen congested.

LITERATURE.

The literature of sarcoma of the stomach may be found in the following references which include the most important contributions to this subject.

Schlesinger. Zeitschrift für klin. Medizin, Bd. 32, Suppl. Heft, 1897, p. 179.

Fenwick. Cancer and Tumors of the Stomach, 1902, pp. 272-283.

Alesandri. Mittheilungen aus dem Grenzgebiet der Med. und Chir., Bd. 12, Heft 4.

Oberst. Beiträge zur klin. Chirurgie, Bd. 45, 1905, Heft 2, p. 477.

DISCUSSION.

DR. HARLOW BROOKS: I have only met with four cases of sarcoma of the stomach in about 1,200 consecutive autopsies. In only one of these cases was the condition made out before death.

DR. A. L. BENEDICT: About a year ago I was called in consultation to see a patient who complained of rather vague stomach symptoms. Several years before the patient had had a tumor of one of the lower ribs which was regarded as sarcomatous, and which had been removed. The question arose whether the stomach symptoms were due to a metastatic growth. The case has survived two years and I would like to ask if this could preclude the existence of gastric sarcoma.

DR. MANGES: There were no metastases in either of my cases, but cases in which secondary sarcomatous growths occur are by no means rare. Not long ago we had a case at Mt. Sinai Hospital in which there was a secondary sarcomatous growth in the stomach following a primary osteo-sarcoma of the jaw that had been removed four years before. The life prognosis in these cases is usually about a year or eighteen months. In one of the two cases of sarcoma of the stomach that I reported, the chemism was exactly the same as in cancer of the stomach; hence the question of the secretion of free hydrochloric acid would not help us much in the differential diagnosis.

[Reprinted from THE MEDICAL NEWS, July 15, 1905.]

THREE ADDITIONAL CASES OF PRIMARY SARCOMA OF THE STOMACH.[1]

BY HARLOW BROOKS, M.D.,

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YOUR president has been kind enough to permit me to report three additional cases of primary sarcoma of the stomach, not as a paper, but as a postlude to the very interesting and instructive study which Dr. Manges has just presented to us. I feel somewhat embarrassed to present these cases to you in this abbreviated and superficial way, for I believe that they and the subject warrant an extended study, but in the few days in which I have had notice of your indulgence I have had time to extract in a very brief way only, the most essential notes on the cases which I hope to discuss more fully at some future time when I shall be better prepared on the subject which Dr. Manges has so opportunely brought to our attention.

Case I.—J. M., female, aged fifty-seven years. The patient entered the hospital in a delirious condition, at times she cleared up somewhat, but was not mentally competent at any time. Friends stated that she had been alcoholic since the age of fifty. Previously she had always been in good health until within the past two months, when she commenced to lose strength and appetite. Her only complaint has been of general weakness. Temperature on admission, 99° F., pulse 86, respiration 24.

The physical examination was practically negative, except for the following conditions—pulse, weak, regular, considerable degree of arterial disease and tension.

I saw the woman several times but was never able to make anything definite out of the case, other than general arteriosclerosis and senility. The extreme emaciation and a peculiar brownish pigmentation of the skin, at one time gave rise to a question of Addison's disease and Dr. Lam-

1 Read before the Gastro-Enterological Society, April 24, 1905.

bert, under whose care she was, insisted throughout that the condition was one of new growth cachexia, though careful examination, particularly of the flaccid abdomen gave no hint as to its location.

Milk only was supplied for nourishment, this was retained and apparently fully digested. No gastric tenderness or tumor was present. Whisky and strychnine were administered from time to time, but the weakness and emaciation steadily increased. After she had been in the hospital fourteen days, she vomited one night, four to six ounces of blood-stained material. This was not saved by the nurse. In the morning a small amount of coffee-ground vomitus was also discharged. The patient at this time was seen to be moribund and she died within a few hours.

Abstract of Protocol.—General appearance—Emaciated and senile. *Heart*—Brown atrophy; aneurismic dilatation of the sinuses of ascending arch. Marked coronary sclerosis; moderate arteriosclerosis of arch of aorta. *Thyroid*—Left lobe contains an encapsulated mass, thought to be an adenoma. *Lungs*—Chronic miliary tuberculosis of apices. *Lymph nodes*—Large, anthracotic and tuberculous. *Liver*—Fatty degeneration and chronic congestion. *Spleen*—Old-healed infarctions. *Pancreas*—Negative. *Stomach*—There is an hour-glass constriction 5 cm. from pyloric ring. Both the upper and lower curvatures of the stomach show thickening of the walls without definitely circumscribed tumor formation. In places the mucous membrane lies free over the surface of the new growth, but in other places the mucous membrane is sloughing and is replaced by a layer of mingled blood clot and necrotic tumor material. The stomach as a whole is small, and except for the ulcerated areas its mucosa is free and anemic. *Intestine*—The intestine is adherent, in a few places, to the peritoneum and in these areas minute tubercles are found, supposed to be recent tubercles, corresponding to these points internally, minute ulcerations of the intestinal mucosa are seen in a few places. These ulcers are thought to be *tuberculous*. *Adrenals*—Negative. *Kidneys*—

Chronic interstitial nephritis. *Genital organs*—Atrophic. *Blood vessels*—The inferior vena cava presents a dilatation filled by recent thrombus located just above the bifurcation. The descending aorta and the large trunks of the abdomen and pelvis show marked arteriosclerosis.

The gross anatomical diagnosis was senility, complicated by gastric carcinoma and adenoma of the thyroid gland with healed pulmonary tuberculosis terminating with thrombosis of the inferior vena cava.

Greatly to my surprise microscopical examination showed that the gastric growth was a small round-cell sarcoma, apparently originating in the stomach wall. The minute nodules found in the peritoneum were also found to be sarcomatous.

Case II.—The case was that of a male, fifty-seven years of age, a baker by occupation. He first came to me complaining of pain just above the umbilicus. His previous and family history were good and gave no hint as to the nature of the present trouble. At times paroxysmal vomiting occurred, usually when the stomach had been overloaded. The vomitus never contained blood.

The man was a large and powerful German, very well nourished. Examination of chest and abdomen gave only negative results, perhaps due to the large amount of abdominal adipose. Hemoglobin, 65 per cent. No hyper-temperature, pulse and respiration normal. He was given a simple rhubarb and soda mixture and was advised as to diet.

Two months later he returned showing marked emaciation, so that palpation of the abdomen was now an easy matter. He still complained of abdominal pain, sometimes very severe and cramp-like in character. He had become very weak, extremely pale and had lost flesh in a truly astonishing manner. He had no desire for food and even fluids were followed by extreme distress.

Palpation of the abdomen disclosed a firm globular mass in the hypochondrium a little to the right of the median line. Inflation of the stomach and ausculatory percussion demonstrated the mass to be apparently attached to the stomach, which was not, however enlarged. Con-

siderable tenderness was present over the mass. Examination of the gastric contents, after a test meal, showed delayed digestion, and apparently more or less pyloric stenosis. Both free hydrochloric acid and lactic acid were found in small amounts. No blood was found and the Boaz-Oppler bacillus was not present. The patient was given a solution of dilute hydrochloric acid and was placed exclusively on a milk-diet. He was seen frequently. Considerable gain of weight and improvement of symptoms was made under this treatment but no decrease in the size of the tumor was observed and he was strongly advised to submit to an exploratory laparotomy.

This was performed at the Hood Wright Hospital by Dr. Lucius Hotchkiss.

The pyloric extremity of the stomach was found to be involved for a distance of about 10 cm., by an infiltrating growth, subperitoneal, but of such extent, that considering the condition of the patient, it was not thought prudent to attempt enucleation or other operative procedure.

The patient died about ten days after the operation and, as we were unable to obtain an autopsy, the house surgeon, Dr. Hanscome, made a thorough exploration of the abdomen through the operation wound and removed portions of the various viscera for examination.

The growth was found to be both submucus and subserous, infiltrating between the walls of the stomach. Microscopical examination demonstrated it to be an angiosarcoma. No metastases were found in any part of the body, though, as I have stated, a complete autopsy was not performed.

Case III.—R. L., female, about fifty years old. Entered Gouverneur Hospital, July 4, 1904, in the service of Drs. Huddleston and Braunlich. Two years ago she had attacks of flatulence, diagnosed as "catarrh of the stomach." There was pain after eating, sour taste in the mouth and similar symptoms. Eight months ago she began to lose flesh and strength. She had occasional attacks of pain over the epigastrium and vomited once in a while, but she had never ejected material resembling blood. On two occasions she was at Mt. Sinai Hospital where a diagnosis of

gastric carcinoma is said to have been made. During this time she twice vomited blood.

The emaciation and asthenia have constantly increased, so that the patient is now bedridden and has developed bed sores. She is extremely anemic, has fever at times and is much troubled with constipation.

Inflation of the stomach demonstrated the lower border to be two fingers breadth above the umbilicus. Although abdominal walls were very thin, owing to the great emaciation; no gastric tumor could be demonstrated and gastric tenderness was doubtful.

The general appearance was that of a woman of medium height, extremely emaciated and of poor muscular development. The skin was very pale and of a waxy appearance. She had an expression of anxiety and suffering and was very apprehensive of being given pain.

Examination of thorax was negative except for marked emaciation. There was slight edema of the left leg, but otherwise the extremities were negative. Examination of the abdomen on many occasions always gave absolutely negative results. Vaginal and rectal examinations were also negative.

Frequent examination of the stomach contents showed the constant presence of free HCl and also of lactic acid. The Boaz-Oppler bacillus was present.

The anemia was profound and the hemoglobin finally sank below 10 per cent., as determined with Dare's instrument; the red-cell count below 1,000,000, and the blood presented the characteristic picture of a very grave secondary anemia. The urine constantly showed the presence of slight traces of albumin and occasionally considerable numbers of casts were found. The weakness, emaciation and anemia constantly progressed in spite of rectal and gastric alimentation and she died, July 16.

A post mortem was absolutely refused and consequently the only possible examination was made per vaginam, but this was done in a quite satisfactory manner, all the thoracic and abdominal viscera being removed and submitted to examination.

The following extracts of the examination touch only on the points of direct interest: "The entire pyloric end of the stomach shows a thickening of the walls, chiefly in the muscular and submucous layers. This thickening is diffuse, not nodular, and is very firm in consistence. The mucosa of the stomach is very anemic and in a few areas minute hemorrhagic extravasations are to be made out. The mucosa moves freely over the submucosa and is apparently not at all involved by the process noted. Although the entire pyloric ring is thickened, it does not amount to a clinical obstruction."

Both gross and microscopical examinations of the other viscera showed only the changes consequent upon severe anemia. The lymph nodes were in particular inspected, but no metastases were found except in certain of the smaller nodes of the gastric mesentery, which showed a few small patches of invasion.

Microscopical study of the growth showed it to be a fibrosarcoma, though in places the diagnosis of myxosarcoma might be granted.

To my mind there is no doubt but that primary sarcoma of the stomach is much more frequent than is generally thought to be the case. Unquestionably this is due to the fact that microscopical examination of tissues removed post mortem is not invariably resorted to. The four recorded by me have been discovered in a series of some 1,200 consecutive autopsies, and in each of these instances the gross specimen aroused my suspicion as to the unusual nature of the growth. It stands to reason that gastric sarcoma may, in many instances, resemble the more common gastric carcinoma, and it is my firm belief, that were every case of stomach neoplasm examined microscopically, we would find sarcomata much more frequent than my statistics indicate.

Judging from my four cases, a very small and insufficient number, it is true, it seems that in several respects, sarcoma of the stomach differs materially from the usual course of gastric cancer.

There is less tendency to involvement of the *gastric mucosa* and more toward infiltration of

the muscular walls. Metastases seem to be less frequently formed and of later appearance and the course of the disease is longer than usual in carcinoma.

It has seemed to me for some time, after a study of my own cases and of such as I have found in the somewhat meager literature of the subject, that it may be possible to make a differential diagnosis in some cases of gastric growth, between cancer and sarcoma of the stomach. Obviously this can be done in but a small number of cases, but in these it is apparently of considerable importance since the sarcomata seem to present a more hopeful field for surgical intervention than the carcinomata on account of the less tendency to general metastasis and invasion by contact.

I feel certain that in the instances cited above the distinction should have been made in Cases II and III particularly, as it was possible to satisfactorily palpate the general abdomen contents in these cases.

The presence of gastric tumor, with both free hydrochloric and lactic acid in the stomach washings, the long course of the disease without discoverable metastasis, with little or no blood in vomitus, even in the later stages of the disease, should, in my opinion, lead at least to the serious consideration of the possibility of gastric sarcoma.

In closing these case reports permit me to say that in my opinion cases of gastric tumor should be submitted to surgical council, even to exploratory laparotomy, just as soon as diagnosis becomes a matter of tolerable certainty.

[Reprinted from THE MEDICAL NEWS, July 15, 1905.]

TWO CASES OF EPILEPSY, THE RESULT OF GASTRO-INTESTINAL DISTURBANCE.*

BY FRANK H. MURDOCH, M.D.,
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REGARDING the pathogenesis of epilepsy a well-known author has recently expressed himself as follows: "For some years the writer has been impressed with the facts forced upon him, not only as the result of the study of his own cases, but by the accumulated researches of Raymond Petit, Voisin, Smith and Herter, Mott and Halliburton, and later by Carlo Besta, and other Italian investigators, that toxemia enters more largely than is generally supposed into the pathogeny of epilepsy, and this is apparent, as Voisin has pointed out, in the general resemblance and mode of occurrence between the epileptic attacks, and certain ordinary forms of poisoning in which the nervous system is affected. In such cases the element of convulsion, and the mental disturbance have features in common, and it would appear that the occurrence of the fit betokens the accumulation in the body of some particular toxic agent, the unwelcome presence of which gives rise to a convulsion when the point of cell-tolerance has been reached. He also states that autotoxemia may occur in three different ways. First, in the existence of a toxemia of gastro-intestinal origin, which was described originally by Bouchard, and later by Herter and Smith, as well as by the author just quoted. Second, by the introduction of certain familiar cocci into the circulation, and third, the presence in the blood of a specific poison called cholin." [1]

The cases which I wish to report were due to autotoxemia, one resulting from disturbance in the stomach, and taking the form of *petit mal,* the other being associated with intestinal disturbance, and taking the form of *grand mal.*

Case I.—Miss M. S., aged eighteen years, consulted me on June 21, 1904, and gave the following history: A year and a half ago she began

* Read at a meeting of the Gastro-Enterological Association, in New York, April 24, 1905.

to be troubled with vomiting during or immediately after meals, occasionally through the day, she suffered from attacks of nausea, and often at such times vomited a very sour fluid. Six months ago she lost consciousness for a few moments while at church, and similar attacks have been coming on with increased frequency ever since, so that for the last two or three months she has had an average of two or three attacks a day, and during the last two days she has had eight or nine attacks each day. There is a distinct aura which begins in both heels and passes up the posterior aspect of the legs, then along the spine, and when it reaches the lower dorsal region she experiences intense nausea, and endeavors to induce vomiting by passing her fingers as far back over the root of the tongue as possible. Simultaneously with the nausea she loses consciousness, for after the attack she has no recollection of having made an attempt to induce vomiting. She looks pale, and has lost twelve pounds in weight. Her menses are scanty and irregular. Appetite poor, bowels regular, bad taste in the morning. Inflation of the stomach shows that its lower border reaches the navel, and the right kidney is movable in the second degree. The gastric secretions are normal. The urine is acid, and contains neither sugar nor albumin. Her stomach in the morning before she has eaten anything, contains a large quantity of mucus mixed with bile. The treatment consisted in proper diet, iron and arsenic, and gastric lavage. It is a remarkable fact that after the first washing out of her stomach the epileptic attacks ceased, and I am glad to say, did not return. She spent the summer in the mountains, being careful as to her diet, taking the iron and arsenic, and washing out her stomach two or three times a week. I saw her on October 11, 1904, and she had gained ten pounds in weight, her menses were regular as to time, and sufficient in quantity, and in fact she was enjoying excellent health.

Case II.—On October 7, 1901, Mr. A. D., aged thirty-two years, merchant tailor, consulted me and gave the following history: Two years ago he began to suffer from frontal headaches.

after the headache had lasted for three or four hours he began to vomit a greenish bitter fluid mixed with food particles. The vomiting continued for twenty-four or forty-eight hours, during which time he had to keep his bed. After the attack he felt quite well for three of four months, then, without any apparent cause, the headache and vomiting would return. Lately the attacks have been coming on every month. On the night of September 1, 1901, he vomited after having retired, but lay down and went to sleep again. Soon after, he woke a young man who happened to be sleeping with him that night, by having a violent convulsion. His friend went for a physician who was of the opinion that the attack was epileptic, as the patient was unconscious, had foam on his lips, and had bitten his tongue. On October 1, during the day, he had headache and vomiting, but no epileptic attack. His appetite is poor, bowels constipated. Examination shows that the apex beat is displaced, being 2½ inches below, and 1 inch within the nipple line; liver normal, stomach and intestines contain much gas. When the stomach is inflated with air the lower border reaches a point 1½ inches below the navel, and the ascending colon is considerably dilated. The gastric secretions are normal, free hydrochloric acid being + 32, total acidity, 68. The urine is acid, specific gravity, 1.020, and contains neither sugar nor albumin, but indican in excess. Treatment consisted in a regulated diet, and flushing the colon every day with normal salt solution. After beginning treatment the patient had no more headache or vomiting, and no further epileptic attacks. Now, it is impossible to tell whether this patient had more than one convulsion or not, for, as is well known, when attacks come on at night a person may be epileptic for years without knowing it.

In regard to diet in epilepsy, Hamilton[2] says, "I have no reason to change my opinion, advanced nearly thirty years ago, and which has been shared by others, that an excessive nitrogenous diet is apt to aggravate the disease. Merson, adopting the idea of Hughlings Jackson, that it is the nitrogen that leads to ex-

plosiveness and instability, conducted a series of experiments at the West Riding Asylum, which showed that a meat diet, not only produced a dull languid condition with an increased temperature, but that the attacks were more frequent." Burney Yeo[3] is of the opinion that epileptics should not have meat more than once a day, and at the Craig Colony for Epileptics in New York, patients are allowed meat once a day every day in the week except Friday.[4]

It has been my experience that patients with achylia gastrica do better on a diet from which red meats have been excluded, while in hyperchlorhydria the reverse obtains. It would be interesting to know the state of the gastric secretions in those epileptics in which the disease is aggravated by a nitrogenous diet.

Of the several forms of diet recommended in epilepsy, Turner[5] has found that of salt starvation the most satisfactory. The chief feature of the method lies in the diminution of the bromide dose, from 20 to 30 grains of one of these salts at bedtime being sufficient.

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[Reprinted from THE MEDICAL NEWS, July 22, 1905.]

ON THE RELATIONS OF SOME OF THE METABOLIC DISEASES TO INTESTINAL DISORDERS.*

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WHEN I was asked to contribute a paper on the relation of certain metabolic diseases to intestinal disorders, I consented to do so with some hesitation. I fully appreciated the fact that the well-being of the individual was intimately connected with the proper carrying on of the normal intestinal functions; also that many of the ailments the human organism is subject to are dependent upon derangements in the intestinal canal. My hesitancy was mainly due to the fact that I realized that but few, if any, of the diseases we are accustomed to designate as metabolic in origin, such as gout, diabetes, etc., are due essentially to disorders in the intestine. On considering the matter, however, I recalled that some of the more obscure and rare metabolic diseases have been attributed by investigators to disturbances in intestinal function. I thought that a review of some of the main facts concerning these diseases, particularly regarding alkaptonuria, ochronosis, and cystinuria, might be of interest to the members of the association.

I. Alkaptonuria and Ochronosis.—Alkaptonuria was first described by Bödeker[1] in 1859. The characteristics of the urine in this condition are briefly as follows: When voided it has a normal appearance, but rapidly acquires a deep-brown color and ultimately becomes black on exposure to the air. The brown color is greatly intensified by the addition of an alkali, its development being accompanied by the absorption of oxygen from the air. The urine is of normal specific gravity.

* Read before the American Gastro-Enterological Association, New York, April 24, 1905.

It reduces alkaline copper sulphate solutions with the aid of heat, the mixture at first being of an inky-black color. Ammoniacal silver nitrate solutions are reduced in the cold. It does not reduce alkaline solutions of bismuth. The urine does not ferment with yeast, and it is optically inactive. The addition of a ferric chloride solution produces a transitory bluish-green color. Diapers and linen moistened with the urine turn a deep brown color on exposure to the air.

Bödeker called the substance which produced these interesting urinary changes alkapton (from "alkali" and "καπτειν," to absorb freely), owing to the property that urine containing it possessed of rapidly absorbing oxygen from the atmosphere in the presence of an alkali. He did not isolate the substance. It was not until 1891 that Baumann and Wolkow[2] demonstrated that the peculiar reactions of alkapton urine were due to the presence of homogentisic acid (Homogentisinsäure, $C_8H_8O_4$ or $C_6H_3(OH)_2.\ CH_2.\ COOH$, which is the dioxyphenylactic acid derived from hydrochinon. This acid has been found in practically all the cases reported subsequent to that date and in the urine of some of the cases previously reported, and is now conceded by all observers to be the cause of the urinary changes in alkaptonuria. The lead salt of the acid, lead homogentisate, is easily obtained by heating the urine nearly to the boiling point and adding five or six grams of neutral lead acetate to each 100 c.c. of urine and allowing it to cool for twenty-four hours. The lead salt crystallizes out in the form of brownish, stellar-shaped, acicular crystals, watery solutions of which give all the reactions of the original urine. The acid itself crystallizes with one molecule of water in large, transparent, prismatic crystals, which become non-transparent at the temperature of the room with the loss of the water of crystallization. They melt at 146.5° to 147° C. They are soluble in water, alcohol and ether, but nearly insoluble in chloroform.

The question now arises, what is the source of the homogentisic acid? It is this side of the consideration of alkaptonuria which brings it into bearing with the subject under discussion. Bau-

mann, Wolkow and Embden, and others, believe that homogentisic acid is derived from tyrosin. Tryosin ($HO.C_6H_4.\ C_2H_3(NH_2).COOH$), or amido-oxyphenylacetic acid, is a normal product of trypsin digestion of proteids in the upper part of the intestinal tract, being formed by a hydrolytic cleavage process. The above-mentioned investigators have shown that when tyrosin is fed by mouth to an alkaptonuric individual nearly the whole of it is excreted as homogentisic acid, whereas the acid is not excreted in the urine when tyrosin is ingested by a healthy person. Beyond the fact that tyrosin and homogentisic acid are both aromatic acids, the two have nothing special in common, although of somewhat similar chemical constitution. The conversion of the former into the latter involves a shifting of the hydroxyl groups of the benzene ring; in other words, a simultaneous oxidation and reduction within the molecule. The view generally held regarding the etiology of alkaptonuria is that originally advanced by Baumann and Wolkow, who thought that the conversion of tyrosin into homogentisic acid is due to bacterial activity, and consequent putrefactive conditions in the upper intestinal tract. They even suggested that there might be a specific bacterium in such cases.

Some clinical facts regarding alkaptonuria may not be without interest. It is congenital in the vast majority if not all of the cases. In a good many of the cases a history is obtained that the individual's urine in infancy stained the napkins a dark-brown color. It persists throughout life. That it is rare is demonstrated by the fact that A. E. Garrod[3] found that only 40 cases had been reported up to 1902. Four of these were reported from the United States, one by myself in 1898. The condition predominates in males. Of the 40 cases, 29 were males and 11 females. The anomaly is likely to make its appearance in two or more brothers and sisters whose parents are normal and among whose forefathers there is no record of its having occurred. There is only one recorded instance of alkaptonuria having been transmitted from an affected parent to a descendant. This occurred in one of two sons of the patient with alkaptonuria I reported.[4]

The investigations of A. E. Garrod have thrown new light on the subject and given us a new conception of the anomaly. As a result of his research he finds that a very large percentage of the cases, concerning which he has been able to collect data, occurred in individuals who are the offspring of the marriage of first cousins, who do not themselves manifest the peculiarity. He points out that in this respect alkaptonuria resembles albinism and possibly also cystinuria. He has advanced the view, therefore, that alkaptonuria is a "sport" or alternative mode of metabolism, congenital in origin and persistent throughout life. In favor of this view is the fact that each individual excretes from day to day a very constant amount of homogentisic acid, the variations being within narrow limits and easily explained by fluctuations in the amount of proteid food taken from day to day. The average daily excretion of the acid has ranged between 2.6 grams in a child three and one-half years old and 5.9 grams in a young man of eighteen years. One would reason, therefore, that alkaptonurics suffer from some inherited metabolic defect, and it seems probable that this defect is closely related to the metabolism of tyrosin. This conception necessarily modifies the view previously held that alkaptonuria results from putrefactive changes in the upper intestinal tract. Being apparently a congenital anomaly, it does not seem possible that it is due to abnormal bacterial activity in the intestine. It does not, however, weaken the view that it is associated with some intestinal digestive disturbance. It would appear that there is some congenital defect in proteid digestion in the intestine whereby homogentisic acid is formed instead of tyrosin.

The general health of alkaptonurics appears to be in no way impaired. Until a very recent date. the only clinical significance that alkaptonuria was thought to possess, rested on the fact that a number of the cases, before recognition, have been mistaken for diabetes mellitus, owing to the power the urine possessed of reducing alkaline copper solutions. The fact that the urine does not ferment, is optically inactive and does not re*duce bismuth,* together with its peculiar color on

standing and on the addition of an alkali, should have obviated such a mistake, however.

Recent observations indicate that a pathologic condition of great interest and rarity is associated with and probably due to the metabolic disturbances in alkaptonuria. This is the remarkable affection known as "ochronosis" or pigmentation of the cartilages, first described by Virchow, in 1866. In Virchow's case there was remarkable pigmentation of the cartilages of the body of a man, aged sixty-seven years, who died of the effects of an aneurism. He gave the name ochronosis to the condition owing to the ochre-like discoloration of the cartilaginous tissues. Osler,[5] one year ago, reported two cases of ochronosis in alkaptonuric brothers, both of which cases had been previously reported in the literature as alkaptonurics, one by Marshall and the other by myself, and constituting two of the four instances of alkaptonuria observed in this country. Previous to Osler's two cases, only seven instances of ochronosis had been recorded. Two of them were described as having "melanuria," but Albrecht,[6] who published the seventh case in 1902, deserves the credit of first suggesting an association between the disease and alkaptonuria. The urine of his patient was dark colored and reduced sulphate of copper solutions, but homogentisic acid was not absolutely demonstrated.

Osler's first case was a man fifty-seven years of age and was the alkaptonuric I reported. The sclerotic coats of the eyes showed areas of pigmentation on the exposed portions. The cartilages of the ears, particularly the concha and helix, presented a remarkable blue-black discoloration. A noteworthy feature not previously recorded was that a butterfly-shaped area of the skin over the nose and cheeks was also remarkably pigmented, the pigmentation very closely resembling powder marks. The discoloration of the sclerotics and of the ears was present, but less marked, eight years ago. There was a very marked bowing of the spine with the convexity backward and he walked with a marked stoop of the shoulders. The patient has been under my own observation for some time and he occasionally has some stiffness of the knee-joints in

which there is marked crepitation on passive movement. The pigmentation of the cartileges of the metacarpo-phalangeal joints has recently been evident through the skin over the knuckles. The other brother, forty-nine years of age, presented a similar but less striking pigmentation of the sclerotics and cartilages of the ears. He had noticed the pigmentation for several years. The patient died of pneumonia but no autopsy could be obtained. There is little doubt but that the deep-seated cartilages of both these cases would have manifested the same changes.

The probabilities, therefore, are that ochronosis is another manifestation of the metabolic disturbances in alkaptonuria, which previously had been recognized only by the urinary changes. There is no definite evidence that the ochronotic condition in any way affects the health or the body functions. It is worthy of note, however, that in one of Osler's cases the joint affection objectively simulated conditions found in arthritis deformans. Some of the cases of ochronosis have not been associated with alkaptonuria and Garrod is inclined to the view that there may be two distinct groups of cases. The interesting fact is that Ogden's alkaptonuria case, another of the four American cases, has recently been found to have the ochronotic pigmentation. Whereas all alkaptonurics do not have ochronosis, it will probably be shown eventually on careful examination that most cases of ochronosis exhibit alkaptonuric changes in the urine.

II. Cystinuria.—Another metabolic affection which has generally been regarded as being dependent on intestinal disturbances is cystinuria, characterized usually by the spontaneous deposition of hexagonal crystals of cystin in the urine. The condition is rare, as shown by the fact that up to November, 1904, Simon and Campbell[7] found that only 131 cases had been reported in the literature. Cystin ($C_6H_{12}N_2S_2O_4$) is the di sulphide of di-amido-ethidenelactic acid. According to Baumann and Goldmann a substance similar to cystin occurs in very small amounts in normal urine. This substance occurs in large quantities in the urine of dogs after poisoning with phosphorus. Although it has not been

definitely demonstrated that cystin is a normal constituent of the urine of human individuals, yet there is good reason to believe that cystin is an intermediate product of normal metabolism and that it is ordinarily broken up with oxidation of the greater part of its sulphur to sulphuric acid. According to one view cystin is apparently preceded in the course of normal metabolism by cysteïn. This cysteïn is regarded as a lactic acid, $CH_3CH.(OH)COOH$, in which one H is replaced by NH_2 and the OH by SH. Cysteïn is therefore to be regarded as an amidothiolactic acid. V. Bergmann, Simon and Campbell, as a result of their researches, are led to conclude that a certain amount of the cystin liberated during normal metabolism is eliminated in the bile in combination with cholalic acid as taurocholic acid, while the remainder appears in the urine in completely oxidized form.

The clinical significance of cystinuria is not great, because it is commonly unassociated with definite symptoms and may be well developed in persons apparently in good health. Occasionally the individuals have disordered digestions, at times associated with anemia. Cystinuria may be of surgical importance owing to the fact that renal calculi composed of cystin may occur. The crystals of cystin when shaken up with urine present a curious sparkling appearance. The urine on decomposing emits a very offensive odor of sulphureted hydrogen, owing to the large amount of sulphur contained in the cystin molecule. Cystinuria is much commoner in males than in females.

What occasions the development of cystinuria? The view usually held is that the condition is closely related to putrefactive changes in the intestinal tract. Probably the most striking feature at present known to us in connection with the pathology of cystinuria is the almost regular association of the condition with the presence in the excreta of putrefactive basic bodies or ptomains belonging to the class of diamines. The diamines are bodies in which two amine (NH_2) groups are united to a diatomic alcohol radical of the olefine series of hydrocarbons. The diamines of greatest in-

terest in connection with cystinuria are *putrescin,* which is tetra-methylen-diamin and *cadaverin,* which is penta-methylen-diamin. These were found in the urine in cystinuria by Baumann and Udránszky as well as in the intestinal contents, while under normal conditions they are not present. Hammarsten thinks that a connection probably exists between the formation of diamines in the intestine, by the peculiar putrefaction in cystinuria, and cystinuria itself. Herter asserts that it is, on the whole, not unlikely that both the diamines formed by putrefaction in the intestine and the cystin formed in the course of metabolism are the expression of an obscure form of putrefaction in the intestine. It must be remembered, however, that a number of competent observers, among them Cammidge and Garrod, have not been able to demonstrate the presence of these diamines in the urine and feces of the cystinuric individuals they have examined.

The recent view of Simon and Campbell must be mentioned here. It will be recalled that in normal metabolism they think that the cystin of the body is eliminated in the bile in combination with cholalic acid as taurocholic acid, while the remainder appears in the urine in completely oxidized form. They found that while the administration of cholalic acid in the non-cystinuric individual leads to a diminished elimination of sulphur in the urine, this does not occur in the cystinuric subject. This suggests to them that in the cystinuric individual taurocholic acid is possibly not formed at all, in which case they are forced to assume either that all the sulphur liberated in the body is eliminated in the urine, or that a portion is excreted in the bile in a form different from taurocholic acid.

It is of interest to point out here that A. E. Garrod advances the view that cystinuria, like alkaptonuria and albinism may, on more thorough investigation, turn out to be an example of a "chemical sport." In this connection he cites a most interesting family described by Pfeiffer, which he thinks supports this view. *Both parents* were normal, but all their four *children, two* daughters and two sons, were cys-

tinuric. The elder daughter had two children, neither of whom was cystinuric.

III. Diabetes and Gout.—In a discussion of this kind it seems advisable to say something of these two most characteristic examples of metabolic diseases, if only to point out how little dependent they are upon disorders in intestinal function. The writer fully appreciates the fact that many gastro-intestinal disorders occur as symptoms of these two diseases, but time will not permit their being considered here.

According to the latest conception of the disease, diabetes results from disturbances in sugar metabolism owing to deficiency in the production of glycolytic bodies formed in the pancreas and muscles (Cohnheim), and which are necessary for the final combustion of the sugars in the latter tissues. It is doubtful whether intestinal disorders in any way, therefore, play any part in the etiology of diabetes mellitus. The "alimentary" glycosurias are not dependent upon defective intestinal function but rather to the sudden ingestion of some form of sugar in such a large quantity that the liver has not time to convert all the glucose into glycogen, thus storing it up for future use. Consequently the excess passes over into the general circulation producing a hyperglycemia and a resultant glycosuria.

It is possible, according to recent researches, that there may be intestinal influences in the production of one of the auto-intoxications occurring in diabetes, namely acetonemia. The work of Geelmuyden, Schwarz, Waldvogel and Magnus-Levy has shown that acetone is formed largely, if not entirely, from fat and not from proteids as was formerly believed. Schwarz found that in severe diabetics the administration of butyric acid caused a marked increase in the acetone production. Blumenthal and Neuberg demonstrated the interesting fact that only the butyric acid given by mouth, and not that given subcutaneously, caused an increase in the acetone output. They think that it is necessary for the butyric acid to first undergo some change in the intestinal canal and that this may be due to bacterial influences.

So far as we are aware at the present time intestinal disorders play no special part in the etiology of gout. We know so little concerning the causes for the marked disturbances in the nitrogen metabolism in gout that we are not warranted in making a dogmatic statement that disorders in the intestinal functions play no part in the etiology of this disease.

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DISCUSSION.

DR. JOHN P. SAWYER, of Cleveland: I venture to speak at this time for a moment upon the matter of the stomach disorders in diabetes. About two years ago I presented before this Association a preliminary report upon this subject, and since then I have enlarged my series of cases, which now numbers twenty-four, in the great majority of which I have observed very striking improvement following treatment directed to the stomach. While I do not now say that the stomach has anything to do with the etiology of diabetes, nevertheless the proper care of the stomach of these patients has a very important bearing on the course of the disease, which fact has hitherto not been sufficiently appreciated. In a large number of cases there is hyperchlohydria and gastric catarrh, without any failure of the motor function, and upon washing out the stomach with an alkaline solution there is a marked reduction in the polyuria, and the thirst is less intense. I have often seen this strikingly illustrated, and have called attenton to it in cases where the glycaemia was still present, but the thirst and polyuria were much reduced. My observations along this line have been confirmed by others, and have been carried on for a sufficient length of time to warrant me in making the assertion that no other measure with which I am acquainted begins to give the relief in diabetes that comes from the use of stomach lavage, particularly when hyperchlohydria is present. Under this method I have seen the urine reduced from ten to twelve quarts daily to two quarts, without any alteration in the ordinary bland diet, or the institution of a strict antidiabetic diet. Those cases in which the disease has existed for a number of years do not respond as promptly to the treatment as the more recent ones. In one case where I have succeeded in getting rid of the sugar by the oats cure, I did not get any result by washing the stomach.

DR. EINHORN: In corroboration of Dr. Sawyer's statements, I would say that I also have observed quite a number of cases of diabetes associated with hyperchlohydria, in whom the uses of alkalies reduced the amount of sugar in the urine. On the other hand, I have also seen diabetes associated with achylia gastrica, and for that reason it is always important in these cases to ascertain the condition of the stomach, so that the diet can be properly regulated.

DR. FUTCHER: I experienced a good deal of difficulty in finding sufficient material to present a paper on the subject assigned to me. A great deal could be said in regard to intestinal disturbances occurring in metabolic diseases, such as gout and diabetes, but I tried to confine myself chiefly to diseases or conditions in which the metabolic disturbances which have been attributed to a primary intestinal disorder. I was very much interested in Dr. Sawyer's experience in conncetion with lavage in the treatment of diabetes.

Extracted from the American Journal of the Medical Sciences, July, 1905

OCCULT BLOOD IN THE FECES AND ITS CLINICAL SIGNIFICANCE.[1]

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During the past few years considerable attention has been paid to the clinical importance of recognizing minute quantities of blood in the discharges of the gastrointestinal tract. The term "occult blood" has been given to these minute hemorrhages, since the amount present is always too small to give the usual macroscopic characteristics of blood in the feces or gastric contents. Moreover, by the time the blood is passed from the body the corpuscles are so broken down that they cannot be recognized with the microscope, and so the tests to be described are intended to furnish a method of detecting these traces of blood chemically.

The importance of occult bleeding has been recognized by various authorities for some time, but no systematic investigation of the clinical value of the sign was undertaken until Boas and Kochmann published their series of investigations in 1901 and 1902.[2]

In 1903 Boas[3] published a second article calling attention to the importance of the test, and expressing some surprise that it had not excited wider interest.

In 1904 Hartmann[4] published what is perhaps the most extensive series of clinical investigations upon the value of occult blood that have yet appeared. He made 1000 tests in 257 cases, including cancer, ulcer, most of the diseases of the gastrointestinal tract, and many other diseases of different portions of the body.

[1] Read before the American Gastroenterological Association, New York, April 24, 1905.

[2] Deutsche medicinische Wochenschrift, 1901, No. 20; Archiv f. Verdauungskrankheiten, 1902, Bd. viii.

[3] Deutsche medicinische Wochenschrift, 1903, No. 47.

[4] Archiv f. Verdauungskrankheiten, 1904, Bd. x. No. 1.

Joachim,[1] Schmilinsky,[2] Schloss,[3] Koziczkowsky,[4] and Petrachi[5] have also contributed to the literature upon the subject.

As far as we can ascertain, the paper by Dr. Steele, published in the *Philadelphia State Medical Journal*, February 1905, is the first reference to a series of observations upon occult bleeding published in America or England.

This would indicate not that the tests are unknown in America, for this is far from the truth, but rather that they have not excited the general attention that their importance would seem to deserve. We therefore take this opportunity to review the significance of occult blood in the stools and the technique of its detection, as well as to put upon record the results of our work upon the subject.

During the past winter we have examined 720 stools for occult blood from 100 patients in the wards of the Presbyterian Hospital. Besides this, we have made several series of observations upon the stools of normal individuals to determine the effect of food and iron upon the test.

We have confined our investigations to the stools almost entirely, and have applied the test only occasionally to the stomach contents. We did this because we were never sure, when the test was positive, that the blood did not come from abrasions caused by the stomach tube. Of course, this objection would not apply to vomitus unless the act of vomiting was very violent.

The second reason for examining the stools, rather than the gastric contents, was the greater ease with which the material to be tested could be obtained. This is an important consideration when information is desired concerning occult bleeding at very frequent intervals, as when the sign is used to control the feeding of a case of gastric ulcer. Of course, a negative test in the stomach contents is conclusive. The danger of error in interpretation of the bleeding is greater when the blood is found in the stools, compared with the gastric contents.

Occult bleeding has in general the same clinical significance as visible hemorrhage, except that its recognition is a much more delicate means of diagnosis because the quantity of blood present is so small.

Practically, however, the presence of occult blood is of decided diagnostic value only in the detection of gastric or duodenal ulcer or gastrointestinal cancer, because it occurs with considerably more regularity and frequency in these two affections than in any other condition of the alimentary tract.

The subject has been developed particularly along these lines, and

[1] Berl. klin. Wochenschrift, May 2, 1904.
[2] Münchener med. Wochenschrift, 1903, No. 49.
[3] Archiv f. Verdauungskrankheiten, 1904, Bd. x.
[4] Deutsche med. Wochenschrift, 1904, No. 33.

most of the work done so far, including our own, has been directed by the desire of obtaining a new and useful aid in the detection of ulcer and cancer. The value of the sign depends, of course, upon the care with which various sources of error are eliminated.

On the other hand, the absence of occult blood is of equal and perhaps greater value in diagnosis than its presence. Here there is no element of possible error, and if blood is not found after a series of examinations in any case, then cancer and ulcer can be excluded.

The presence of occult blood will be of value in the diagnosis of carcinoma and gastric ulcer only when sources of bleeding, that have no significance, are excluded. Since the test is very sensitive, very small amounts of blood can be detected, and naturally the liability to error in determining the origin of the hemorrhage must be greater than in large and visible hemorrhages, where the condition of the blood may indicate whether it comes from high up or low down in the gastrointestinal tract. Thus, as has been said, various foodstuffs containing hæmoglobin, if present in large enough amounts, will give the reaction, and blood in the stools may occur after epistaxis, hemoptysis, and in cases of hemorrhoids. Consequently, an important part of the study of occult blood should consist of experiments to determine in what conditions the reaction may occur, without significance in the diagnosis, and a good part of our work has been along these lines.

The two reactions which have been employed oftenest in the investigations into the significance of occult bleeding are the guaiac-turpentine test, made with an acetic acid ethereal extract of the feces as suggested by Weber,[1] and the aloin-turpentine test, made with the same extract as devised by Klunge and Schaer. These reactions are simple, and no especial apparatus is required.

In our experience the aloin reaction has proved the more reliable, and less liable to be obscured by urobilin in the ethereal extract., and we therefore think that it is the more dependable test. However, the guaiac reaction in our hands has given very satisfactory results; indeed, almost as satisfactory as the aloin. We employed both tests upon every stool and seldom found one positive without the other, but we never considered that blood was present unless we obtained a clear reaction with aloin. Our material was obtained from bed patients, over whose diet we had perfect control, and it is to this fact that we attribute the satisfactory results that the guaiac test gave us, for in our investigation upon the effect of diet upon the stools we noticed that the guaiac test responded rather more quickly to increase of rare meats, and that a positive result was often obtained with it when the aloin reaction was negative.

The guaiac reaction has certain disadvantages that prevent it being a perfectly satisfactory test for very small amounts of blood

[1] Berliner klinische Wochenschrift. 1893. No. 19.

in the feces. When a fair amount of blood is present the reaction is clear and the color is a true blue, but when the quantity of blood approaches the extreme limit of the test's power of detection, then the color becomes a green, and then brownish-red, deep brownish-red, or violet as the blood increases in amount until the color scale reaches the blue again. There appear to be some differences of opinion as to the point where the color of a positive reaction merges with that of a negative reaction, and various observers differ as to the border-line. A further disadvantage is that the green and browns are often obscured by the brownish tint of the ether acetic-acid extract, due to extraction of the urobilin.

Weber, in his original article upon the detection of blood in the stools,[1] found that the spectrum analysis of an acetic acid ethereal extract of feces containing blood showed hæmatin; consequently it is reasonable to suppose that the substance in the ethereal acetic acid extract which gives a reaction for blood is derived from the hæmoglobin, which has been changed by digestion into hæmatin.

Deen and later Brandenburg[2] have shown that pus in the urine will turn a fresh tincture of guiac blue without the addition of turpentine or hydrogen peroxide, and this reaction has been used to detect pus in the urine under the name of Deen's test.

Brandenburg found that the constituent of the pus which produces this blue color was a nucleoalbumin, which he precipitated by strong acetic acid from a chloroform extract of the pus. He concluded in a series of experiments that this nucleoalbumin was derived from the leukocytes and not from the tissue cells found in the urine.

Pus in the stools ought not, then, to be a source of error in the reaction for occult blood, because the tests are made with an ethereal extract, made after the feces have been mixed with acetic acid, which should precipitate this nucleoalbumin that causes a reaction similar to that of blood.

It is hard to demonstrate experimentally that pus will not give the test, because it is difficult to obtain pus which is perfectly free from blood. We have tested three specimens of urine containing much pus, and three specimens of pus which were obtained in such a way that they contained very few blood cells, as demonstrated by the microscope, with the following result: an acetic acid ethereal extract of pure pus from the urine does not give the reaction for blood. A specimen of pus containing a very small amount of blood gave only a very slight reaction by the aloin and guaiac tests, which might be explained by the red blood cells present. However, if the substance responsible for the reaction was derived from the leukocytes of the blood and not from the hæmoglobin, then with pus the reaction should have been very intense indeed, which was not the

case. All of the preceding indicates that if the feces are thoroughly mixed with acetic acid before the ethereal extract is used, pus will not be a source of error in the detection of occult blood.

All observers, from Boas to Koziczkowsky, recommend the aloin test as the more reliable and delicate. It has the advantage that it is not as much obscured by bile pigments or chlorophyll in the ethereal extract, and, as has been said, it is extremely delicate. Koziczkowsky experimentally found that the reaction demonstrated 0.25 grm. of blood in 2 grm. of feces.

He has noticed that the liability to error in the color reaction due to the presence of considerable urobilin or chlorophyll in the ethereal extract is much increased by artificial light, and says that the test should always be done in daylight.

He also found that carmine in the stool gave a red color in the reaction almost exactly like the color of the blood reaction. Consequently, charcoal should be used instead of carmine to mark a test meal if this is employed.

Technique of the Test. If the stools are not in a semi-liquid condition they must be made so by thoroughly mixing them with distilled water. We usually employed 5 grm. of fecal matter in every test. After the material has been thoroughly softened the feces must then be thoroughly mixed with at least its own bulk of ether, and the whole well shaken. This is a very necessary part of the procedure, as it removes the fat, which otherwise produces a thick emulsion when the stools are extracted with acetic acid and ether, and renders it almost impossible to obtain a satisfactory ethereal extract. After being thoroughly shaken the mixture of feces and ether should be allowed to stand for fifteen minutes or longer and the supernatant liquor is then poured off. The remaining fecal matter is then mixed with one-third its volume of glacial acetic acid and 10 c.c. of ether. The mixture is again thoroughly shaken and allowed to stand for at least fifteen minutes. The ethereal extract will rise to the top in a clear layer and can be readily poured off.

The solution of aloin used is made by dissolving as much aloin as will go on the end of a spatula in one-third of a test tube of 70 per cent. alcohol; 2 or 3 c.c. of the clear yellow aloin solution are then mixed in the test tube with about the same amount of the ethereal acetic acid extract; 2 or 3 c.c. of ozonized turpentine are then added and the whole is gently shaken. If blood is present the reaction may appear in one of several ways: either the whole mixture turns a pink which gradually deepens to a cherry red; second, or the solution of aloin sinks to the bottom and forms a layer beneath the mixture of ether and turpentine, and this lower layer of aloin in positive tests gradually becomes a deep cherry red. Sometimes if the ether and turpentine are first mixed and then the aloin allowed to flow gently down the side of the tube, the two

of fluids will remain separate and a deep-red ring will form at their junction. Not more than fifteen minutes should be allowed for the red color to show itself, for after this the aloin will gradually turn red, even if blood is not present. It is extremely important to make up the solution of aloin freshly, for when it stands exposed to light it changes into about the color that it attains in the reaction when blood is present. When the test is negative the color remains a light yellow, which becomes a red after standing for some length of time. Hydrogen peroxide does not work satisfactorily as a substitute for turpentine in the aloin test.

The ozonized oil of turpentine should be prepared by allowing a chemically pure oil of turpentine, such as that prepared by Merck, to stand exposed to the air for at least three weeks.

If the guaiac test is to be used, a solution of guaiac must be made by shaking a gram or so of gum guaiac in a test tube half full of ether and allowing the mixture to stand until it becomes clear by settling. A couple of cubic centimetres of this guaiac mixture is mixed with the same quantity of ethereal extract of the feces, at least an equal amount of hydrogen peroxide is added, and the whole is shaken in a test tube. The hydrogen peroxide quickly settles to the bottom and the ethereal extract floats on top. The blue color of a positive reaction shows itself very quickly in the supernatant layer, which in a decided reaction becomes a deep blue, that may be somewhat masked by the brown color of the urobilin in the ethereal extract. In such a case the blue color often becomes a purplish-brown, but even this reaction is mistakable. If the reaction is negative no color change occurs.

The hydrogen peroxide used by us was full-strength Oakland "Dioxygen." Ozonized turpentine makes an oxidizing agent equally as delicate as hydrogen peroxide, and may be used instead of it. When this is done the turpentine is added to the ethereal extract and guaiac mixture and becomes intimately mixed with it, and the whole mixture then gradually turns blue. We have found that the guaiac test is made rather more easily by the use of hydrogen peroxide. The solution of guaiac should be prepared freshly, but not quite as often as the aloin solution.

A series of observations upon two normal subjects has convinced us that rare red meats, if taken in sufficient quantities, will give a very decided reaction for blood in the feces. We made no effort to determine the exact amount of rare meat required to give the test, since we think that there are other elements involved than the mere amount ingested. Thus the speed with which the food passes through the gastrointestinal tract seems to influence the intensity of the test, for the blood reaction appeared rather more intensely after a diet of red meat when a cathartic was taken, probably because insufficient time for digestion was given. Any circumstance that

fibre would favor the finding of blood after red meats. The important thing, then, is not so much the amount ingested, but that red meats will give the reaction, and should be excluded from the diet before the test is made.

We found that two meals daily of rare beef or chops will invariably give the reaction, and one meal may do it. Well-cooked fowl, ham, kidney, and fish never gave a positive reaction. Our results showed that the ordinary winter vegetables were not liable to give a positive test.

Our patients whose stools were tested were always either on "liquid diet," including milk and broths, or on "semiliquid," of milk, eggs, and toast. A few were upon "selected" diet which contained no red meats and only one vegetable. Consequently we can dismiss the question of diet in discussing the results in our series.

We found in our work in the wards that pressed beef-juice, namely, the juice from rare beef, gave a strong positive reaction, invariably within twenty-four hours of the ingestion.

Therefore, before a positive test can be considered of any value, red meats and beef-juice should be cut out of the patient's diet for at least twenty-four hours before the tests are made. It would probably be safer to give a still longer interval, or to mark off the meat-free diet with charcoal and not to make the test until the charcoal appears in the stool.

The test for occult blood must often be made while the patient is upon rectal feeding. Meat extracts occasionally are used in nutritive enemata, and we have shown that some of these meat preparations will give the reaction for occult blood. Therefore, when the stools contain portions of these enemata, the particular beef extract employed should be tested to see whether or not it gives the reaction for blood.

The question as to whether various preparations of iron taken by the mouth will give a reaction in the stools similar to that of blood is an important question, since iron will be very often given in gastric ulcer or other anæmic conditions while the stools are being tested. We have examined the stools of patients who were taking various iron preparations by the mouth. The forms of iron employed were ferric carbonate, 30 grains a day; Blaud's pill, 30 grains a day; Ovoferrin, 1½ oz. daily; Peptomangan, 1½ oz. daily. These preparations were taken for at least one week, and never during their administration did we obtain a reaction similar to that of blood in the stools.

We think that our results apply equally well to all other preparations of iron, since the iron in the stools usually has been changed into a sulphide by the time it reaches the rectum, and the stools that we tested were always greenish-black.

Our investigations seem to show that the ingestion of iron need *not be considered in* examining the stools for occult blood.

The sources of error in the correct interpretation of the presence of occult blood in the stools in the diagnosis of cancer and gastric ulcer arise in the difficulty of excluding all other sources of small hemorrhages. These are many in number and variety. It is usually possible to exclude the conditions that would produce such uncertainty, and it is often worth while to go to considerable trouble to do so, as in the use of the proctoscope, etc. Tuberculous ulcer, typhoid fever, hemorrhoids, fissure, or purpura can usually be excluded with more or less certainty.

On the other hand, it will often be impossible to eliminate conditions that might give rise to error, and sometimes one cannot be certain whether the blood comes from a gastric ulcer or gastrointestinal cancer, or is a symptom of some other affection that it will be impossible, at the time, to exclude. An example of the last condition is cirrhosis of the liver, of which our list contains two cases, both with blood in the stools. One of these was a perfectly typical case in the advanced stage, with small liver, large spleen, and ascites, and we were never at a loss to explain the blood, which probably came from the œsophagus. In the other case (No. 2) operation disclosed duodenal ulcer and in addition cirrhosis of the liver. The symptoms were fairly typical of ulcer, namely: hyperacidity, anæmia, rather indefinite epigastric pain, and occult blood in the stools. There was no jaundice, ascites, hemorrhoids, or other decided signs of portal obstruction. We found, however, a typical small, rough, cirrhotic liver, with exceptionally good collateral circulation through the deep veins of the abdominal wall, which explained the absence of indications of portal obstruction. We were impressed by the fact that all of the symptoms could have been produced by the cirrhosis alone without the ulcer, including the occult bleeding, and we are inclined to think that cirrhosis of the liver will be one of the conditions most liable to cause error in the interpretation of occult bleeding, because it so often exists without symptoms, or with very indefinite symptoms of gastrointestinal disorder, and, besides, shows occult bleeding in the stool quite regularly and frequently.

We have arranged a table of the conditions which might give rise to confusion. We appreciate the fact that the list is incomplete, but it represents our own experience and conclusions.

Extraneous: Red meats, carmine, swallowed blood from any cause, hæmoptysis, epistaxis, and menstruation.

Arising from gastrointestinal tract: Cirrhosis of liver, purpura of gastrointestinal tract, tuberculous ulcer, typhoid ulcer, hæmophilia, hemorrhoids, and fissure and fistula of rectum.

Gastric and Duodenal Ulcer. Our results with the test in gastric ulcer agree in general with those of other observers. We have had the opportunity of studying 9 cases of ulcer of the stomach or duodenum, and have tested, in all, 122 stools from these patients

and in the others the classical symptoms, pain, vomiting, and hemorrhage from the stomach, were present to a greater or less degree in every one.

Occult blood is found intermittently in gastric and duodenal ulcer, thus differing from carcinoma, in which blood appears much more regularly. This, however, is not an invariable rule, and cannot be used as a means of diagnosis between ulcer and carcinoma.

In one of our cases of ulcer blood was present in every stool until it gradually disappeared and the patient recovered entirely, thus excluding carcinoma.

More commonly, however, blood is present at intervals of two or more days. Sometimes only once a week. It is usually more marked after an attack of pain and vomiting, especially before the patient is put upon an ulcer diet.

When rectal feeding is commenced, or nothing but milk is given by the mouth, the bleeding usually stops in a few days. Recurrence of bleeding beyond this time with a milk diet suggests development of cancer in the floor of an ulcer, especially if the patient has reached or passed middle life.

No arbitrary limit can be set upon the time when persistence of bleeding indicates carcinoma, In Case II. blood persisted for eight days on rectal feeding, although there was no suspicion of cancer.

The test for occult blood is a very useful check in the treatment of ulcer. It has been our custom to keep our patients upon rectal feeding until there has been no blood in the stools for a week, and then gradually institute feeding by the mouth, watching its effect by testing the stools for blood.

The test has enabled us to demonstrate the value of milk diet in ulcer by the quick relief it affords from bleeding.

The presence or absence of blood can be used to test the effect of various remedies upon the ulcer. Thus, Schloss showed that the bismuth cure was not a specific remedy as claimed by Fleiner.

Constant recurrence of hemorrhage upon the resumption of solid food would tend to show that the ulcer was a chronic one, and would be an indication for surgical interference as in Case VII.

As has been said, recurrence of bleeding in a milk diet would suggest carcinomatous ulcer.

CASE. I.—Male aged fifty-five years; duodenal ulcer; recurrence. In April, 1903, had tarry stools, no vomiting, hyperacidity, epigastric pain and quick loss of weight and strength. Recovered with rest and liquid diet.

October, 1904. Recurrence of pain in epigastrium, circling around to back; vomiting; rapid loss of weight and strength; vertigo.

Occult blood,	October 18.	Positive.	November 1.	Negative.
Modified ulcer,	October 19.	Negative.	November 4.	Negative.
Diet, no red meat,	October 22.	Negative.	November 16.	Negative.
	October 25.	Positive.	December 8.	Negative.
	October 29.	Intense.	April 11.	Negative.

Recovered weight and strength under treatment, and pain disappeared; recovery.

The recurrence of bleeding and the previous history led to a diagnosis of ulcer, probably duodenal.

CASE II.—Female aged seventy-four years; chronic gastric ulcer recurring attacks of epigastric pain for twenty years; several attacks of vomiting of blood in past year; large hemorrhage from stomach February 6th; intense anæmia; hæmoglobin, 30 per cent.; rectal feeding; recovery.

Occult blood,	February 8.	Intense.	Liquid diet,	February 15.	Positive.
Rectal feeding,	February 9.	Intense.	Iron,	February 16.	Negative.
" "	February 11.	Intense.	"	February 18.	Negative.
" "	February 12.	Positive.	"	February 20.	Negative.
" "	February 13.	Positive.	"	February 22.	Negative.
" "	February 14.	Positive.	"	February 24.	Negative.

Here the gradual disappearance of blood excluded carcinoma. Examination of the stools was a valuable aid in the treatment of the case, justifying the return to mouth feeding. The blood was found in every stool and not intermittently, as is usual in ulcer.

CASE III.—Female, aged forty years; gastric ulcer; four attacks of bloody vomiting in past four months; no pain; extreme anæmia; hæmoglobin, 30 per cent.; epigastric pain.

Occult blood, Rectal feeding, Carbonate of iron in powder,	March 9.	Negative.	Hæmoglobin, 35 %	March 21.	Intense.
				March 22.	Negative.
				March 25.	Intense.
				March 27.	Negative.
"	March 10.	Negative.		March 28.	Negative.
"	March 11.	Moderate.		March 29.	Negative.
"	March 14.	Negative.		March 30.	Negative.
"	March 14.	Negative.		April 3.	Intense.
"	March 15.	Intense.		April 6.	Negative.
"	March 16.	Moderate.		April 11.	Negative.
"	March 17.	Negative.		April 15.	Negative.
"	March 17.	Negative.		April 15.	Negative.
Milk and eggs by mouth,	March 19.	Moderate.	Hæmoglobin, 55 %	April 18.	Negative.
	March 20.	Intense.		April 20,	Negative.

CASE IV.—Male, aged thirty-eight years; duodenal ulcer; pain in epigastrium, radiating along right costal margin to lumbar region; vomiting of tarry material; tarry stools; tenderness in line of ninth rib one inch below costal margin on the right; pain promptly relieved by orthoform; hyperacidity, T. A. 100; free HCl, 60; hæmoglobin, 23 per cent.

Operation confirmed diagnosis and showed ulcer of duodenum and advanced atrophic cirrhosis of liver; gastroenterostomy; death.

Occult blood,	December 20.	Intense.	Milk diet,	December 23.	Intense.
Milk diet,	December 21.	Intense.	"	December 23.	Operation.

The presence of occult blood led to a diagnosis of ulcer. Continuance of bleeding and failure to improve led to an exploratory operation. Blood was found in every stool tested.

CASE V.—Female, aged fifty-six years; chronic gastric ulcer; epigastric pain and resistance; hyperacidity, T. A. 70; free HCl, 40; loss of flesh and strength; hæmoglobin, 80 per cent.; great improvement in flesh and strength.

Occult blood	January 7.	Slight.
Liquid diet	January 8.	Moderate.
"	January 13.	Intense.

A previous history suggesting ulcer; hyperacidity, pain, and blood in the stools pointed to gastric ulcer.

CASE VI.—Female, aged twenty-four years; gastric ulcer; hæmatemesis; pain after food; tarry stools in early stages; anæmia; hæmoglobin, 35 per cent.; milk diet; admitted September 15, 1904.

Occult blood,	September 20.	Positive.	October	7.	Positive.
	September 22.	Negative.	October	10.	Positive.
	October 4.	Positive.	October	16.	Positive.

A typical clinical picture of ulcer.

CASE VII.—Female, aged twenty-three years; chronic gastric ulcer; recurring attacks of bloody vomiting for ten months; pain in epigastrium; secondary anæmia.

Occult blood	September 28, 1904.	Positive.
	October 4.	Positive.
February 4 to March 20, 1905, seventeen tests in all		Negative.
April 2 (after vomiting)		Moderate.
April 5,		Trace.
April 6 to 16 (four tests)		Negative.

Chronic gastric ulcer with hemorrhage from stomach and occult blood in feces whenever solid food is resumed.

CASE VIII.—Female, aged fifty-six years; recurring gastric ulcer; pain in epigastrium; vomiting of blood six weeks before coming under observation; much anæmia; hæmoglobin, 50 per cent.; vomit sour and burning.

Occult blood.		
Milk diet	July 12.	Negative.
"	July 25.	Negative.

Improved greatly by rest and diet; hæmoglobin rose to normal; pain disappeared.

February, 28th. Return of pain, restlessness, and nausea.

Occult blood,	February 28.	Positive.	March	7.	Negative.
Milk diet,	March 3.	Positive.	March	14.	Negative.
	March 5.	Negative.	March	24.	Negative.

Gastric ulcer which had healed when the patient first came under observation; recurrence of pain and nausea led to an examination of the stools, which showed occult blood. With rest and liquid diet the bleeding stopped promptly.

CASE IX.—Female, aged thirty-five years; probable gastric ulcer; nausea; tenderness in epigastrium; much vomiting; much improvement under rest treatment: no hemorrhoids.

Occult blood.			Liquid diet,	October	13.	Moderate.
Liquid diet,	September 26,	Intense.	"	October	14.	Trace.
"	September 27,	Moderate.	"	October	16.	Moderate.
"	September 28,	Negative.	"	October	18.	Moderate.
"	September 29,	Negative.	"	October	19.	Moderate.
"	September 29,	Trace.	"	October	21.	Moderate.
"	September 30,	Intense.	"	October	22.	Intense.
"	October 2,	Moderate.	"	October	23.	Intense.
"	October 3,	Moderate.	"	October	24.	Intense.
"	October 4,	Moderate.	"	October	26.	Slight.
"	October 6,	Intense.	"	October	31.	Slight.
"	October 7,	Negative.	"	November	6.	Moderate.
"	October 8,	Negative.	"	November	8.	Moderate.
"	October 10,	Moderate.	"	November	13.	Negative.
"	October 11,	Moderate.	"	November	14.	Negative.
"	October 12,	Moderate.	"	November	16.	Negative.

A somewhat doubtful case, but probably gastric ulcer. All insignificant causes of bleeding were excluded as far as possible. The great improvement excluded carcinomatous ulcer.

Carcinoma of the Gastrointestinal Tract. We have examined eleven stools in six cases and found blood in every one. This agrees with the larger experience of the various German observers, all of whom unite in the statement that every stool contains blood in carcinoma of any portion of the gastrointestinal tract, provided the tumor has progressed to the stage of ulceration. Consequently the presence of occult blood is of no greater assistance in making an early diagnosis of cancer than are the other laboratory methods; and since occult bleeding does not commence until the tumor begins to soften and ulcerate, the test is not of the same value in determining the time for successful surgical interference as it is in ulcer.

The usefulness of the reaction is greater in excluding cancer than in demonstrating its presence and it loses nothing in importance from this fact. The two conditions in which it is likely to prove of greatest value are in differentiating between gastritis with anacidity and early carcinoma ventriculi without palpable tumor, and between simple ulcer and carcinomatous degeneration of an ulcer.

According to Hartman, the test is of value, in the diagnosis of carcinoma of the pylorus without a palpable tumor, *only* when it is negative, since blood also occurs in the stools of cases with benign stenosis of the pylorus associated with stagnation. But when there is good motility and no pyloric obstructions, and at the same time free HCl is absent, then the presence of blood in the stools is almost a sure sign of cancer.

Our series consisted of—

Carcinoma of the cardia	1 case.
Carcinoma of the liver and pancreas, secondary involvement of the bowels	1 "
Carcinoma of the ileocæcal valve	1 "
Carcinoma of the sigmoid flexure	1 "
Carcinoma of greater curvature	1 "

CIRRHOSIS OF THE LIVER. The following case shows that occult blood may be present in the stools in this disease. The blood may come, of course, from hemorrhoids, which were excluded in this case, or from a dilated œsophageal plexus. It is of no particular diagnostic value; indeed, it may lead to error in diagnosis.

CASE X.—Male, aged forty-six years; white; atrophic cirrhosis of liver; enlarged liver gradually shrinking, slight jaundice; ascites; urobilinuria; frequent nose-bleed and hemorrhage from bowel, but none in the time that the tests were made; severe anæmia; hæmoglobin, 35 to 40 per cent.; occult blood, September 20, 1904, to January 24, 1905; 6 tests, 4 positive.

Case IV. was also an instance of atrophic hepatic cirrhosis.

PURPURA. The stools of one such case contained occult blood, which probably came from purpura of the gastrointestinal mucous membrane.

CASE XI.—Male, aged thirty-seven years; white; malignant endocarditis; jaundice; purpuric spots on the skin; stools sometimes contained visible blood.

Autopsy. Mitral endocarditis; abscess of spleen; infarcts of kidneys; gastrointestinal tract congested; no ulcer; occult blood, September 28, 1904, to October 9, 1905; 5 tests, all positive.

TUBERCULOUS ENTEROCOLITIS. Occult blood was found in the stools of one case. It was associated with visible hemorrhage.

CASE XII.—Male, aged twenty-eight years; white; tuberculous pleurisy and enterocolitis; stools contained mucous and sometimes visible blood; occult blood, December 2, 1904, to December 28, 1905; 10 tests, all positive.

The following table contains the cases of gastrointestinal diseases tested for occult blood, and in which none was found. The list calls for no especial comment. Hartman and others state that benign or spastic pyloric stenosis with stagnation gives the test. We have had no case of this sort and cannot give any data upon the subject.

		No. tests.	Result.	Total acidity. Free HCl.
Case 13.	Gastric neurosis	2	Negative.	40–30
" 14.	" " anacidity	8	"	10–0
" 15.	" " anacidity	1	"	10–0
" 16.	" " heterochylia	11	"	10–0 43–16
" 17.	" " heterochylia	5	"	60–0 60–47
" 18.	Anacidity and anæmia	28	"	8–0
" 19.	Constipation and anacidity	5	"	12–0
" 20.	" " subacidity	2	"	46–0
" 21.	Chronic constipation	1	"	
" 22.	Mucous colitis	1	"	
" 23.	Cholecystitis and hyperacidity	1	"	85–60
" 24.	Tertiary syphilis and subacidity	3	"	50–0
" 25.	*Sarcoma of back*, jaundice, cholecystitis (?)	6	"	44–20
" 26.	*Neurasthenia*; achylia complete, with considerable abdominal pain	6	"	0–0

TYPHOID FEVER. It would be natural to expect occult bleeding in typhoid fever, especially in the second and third weeks, when the ulcers are freshest and more liable to hemorrhage, and we were surprised at the small proportion of positive reactions obtained in our series of observations.

We tested 92 stools in 19 cases of typhoid and found occult blood once each in three different patients—none of whom had visible hemorrhage while under observation. Consequently we think that these three cases were examples of true occult bleeding in typhoid fever.

The first reaction occurred upon the eighth day of a mild case, the second upon the thirteenth day in a severe case, and the third upon the fourteenth day in a moderate case—that is, all in the second week, as would be expected from our knowledge of the pathology of the disease.

The stools were always examined in the febrile stage, and 80 of our 92 tests were made in the interval from the second to the fourth week, when the ulcers should be freshest and more liable to bleed. We have no data as to the stools in the first week, as none of our patients were admitted until the beginning of the second.

The weeks of the disease in which the tests were made are as follows:

Second week	22 tests,	2 positive.
Third "	28 "	1 "
Fourth "	30 "	all negative.
Fifth "	8 "	" "
Sixth "	4 "	" "

The cases may be tabulated in accordance with their severity as follows:

Severe	7 cases,	1 positive test.
Moderate	8 "	1 " "
Mild	4 "	1 " "

Since we commenced our investigations Petrachi has published the results of a systematic examination of the stools in 18 cases of typhoid fever. His proportion of positive cases is much greater than ours, for in 8 of his patients he found occult blood 42 times, independent of visible hemorrhage. Most of his tests were made in the second and third weeks, as in our series, and the proportion of severe, moderate, and mild cases in our two lists are strikingly alike, yet the results of the two series of observations differ radically; for while we found one positive reaction in each grade (or 3.5 per cent. of our cases), Petrachi found 42 positive tests in 44 *per cent. of his* cases, the majority being of the severe and moderate *types.*

We cannot explain the lack of agreement in the two series of

similar in severity, and the stools were examined at the same period of the disease.

Petrachi's table of severity was as follows:

Severe	6 cases,	4 positive tests.
Moderate	8 "	4 " "
Mild	4 "	all negative.

We began our observations with the hope that it might be possible to foretell the occurrence of visible hemorrhage by demonstrating occult blood in the stools some time before visible bleeding appeared. The importance of this is very apparent, since it might be possible to lessen or prevent visible hemorrhage by modifying the treatment as soon as occult bleeding indicated that a large hemorrhage was about to occur. In this we were disappointed, although our investigations have not been extensive enough to be conclusive. In 4 of our cases visible hemorrhage occurred while the patient was under observation. In 2 of them the stools were examined systematically before the hemorrhage occurred, always with a negative result. In the other 2 cases the hemorrhage occurred so soon after admission that a careful study of the stools could not be made.

It will be seen that a prehemorrhagic stage was not demonstrated and no warning of large hemorrhage was given by the presence of occult blood.

Petrachi, on the other hand, found occult blood from one to five days before visible blood appeared in the stool, and he thinks that in this way he can foretell the occurrence of large hemorrhages.

Neither series of observations is large enough to be conclusive, and the subject needs further investigation.

Pernicious Anæmia. We examined the stools in one case, with a negative result.

The absence of blood was a valuable point in the differential diagnosis from carcinoma of the gastrointestinal tract.

CASE XXVIII.—Male, aged fifty years; white; hæmoglobin, 18 per cent.; red cells, 900,000; nucleated reds; achylia gastrica; sometimes slight jaundice; urobilin present; no intestinal parasites, and no visceral lesion to explain the anæmia; occult blood, January 5, 1905, to February 9, 1905; 14 tests, all negative.

The patient developed slight ataxia with absent knee-jerks, then he rapidly improved until his hæmoglobin reached 65 per cent. Subsequently he relapsed, with sudden loss of hæmoglobin, which fell to 30 per cent. Still under observation.

PNEUMONIA. We made 34 tests in 12 cases. Twice we obtained positive results in 2 different patients in the early stages of the disease. The rest of the tests were all negative. We attributed the presence of occult blood in the positive cases to the blood contained in sputum that was accidentally swallowed.

OTHER DISEASES. We tested 33 stools of 11 cases of the following diseases, *always with a negative result:*

Acute mania	1 test.	Negative.
Dementia	4 tests.	"
Phthisis pulmonalis, 2 cases	10 "	"
Ulcerative endocarditis	1 test.	"
Influenza, 2 cases	4 tests.	"
Diabetes mellitus	1 test.	"
Spinal syphilis	2 tests.	"
Senility	1 test.	"
Herpes zoster	1 "	"
Anæmia secondary to malnutrition	8 tests.	"

Classification of Diseases in Relation to Occult Blood in the Feces.

Positive Reaction. Cancer of gastrointestinal tract; gastric and duodenal ulcer; benign pyloric stenosis with stagnation (Hartman); cirrhosis of liver; purpura; tuberculous enteritis; typhoid fever (infrequently).

Negative. Gastrointestinal Diseases. Gastric neurosis with anacidity, subacidity, hyperacidity, heterochylia, or normal secretion; chronic constipation; mucous colitis; cholecystitis and hyperacidity; achylia gastrica; chronic gastritis, with anacidity or normal acidity (Hartman).

Negative. Miscellaneous Diseases. Pernicious anæmia; severe anæmia secondary to malnutrition; phthisis pulmonalis; ulcerative endocarditis; pneumonia; influenza; acute mania; diabetes mellitus; herpes zoster.

Reprinted from the New York Medical Journal for March 10, 1906.

THE DETERMINATION OF THE GASTRIC AREA, WITH SPECIAL REFERENCE TO TRANSPOSITION OF VISCERA, HOURGLASS STOMACH, GASTROPTOSIS, ETC.

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The gastric area is determined to a large degree by ordinary percussion, occasionally by palpation, as when the wall is thickened or when the abdominal wall is soft and thin or when the true abdominal wall is displaced by a large umbilical hernia or by diastasis of the recti. Succussion, which is really a form of palpation, is also employed, especially when the stomach is dilated and partly filled with liquid and gas. In succussion, we palpate not so much the stomach as its contents, and succussion is really a form of ballottement, familiar in the diagnosis of pregnancy, of ecchinococcus cyst with loose daughter cysts, and of certain forms of cholecystitis. Succussion is, however, liable to give confusing results, as we may fail to discriminate between stomach and colon, and may infer too little or too great degrees of dilatation.

Inspection of the stomach, or rather of the area of gastric peristalsis, is feasible if the abdominal wall is extremely thin and, in an indirect way, under normal conditions. Dr. Mark I. Knapp has called attention to the feasibility of determining the lower level of the gastric peristalsis by sighting very obliquely over the abdomen, while the patient

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breathes. I have frequently verified the truth of this statement by other means. Auscultation, especially of deglutition murmurs, may also furnish very helpful hints as to the location of the stomach. Since 1893 I have relied mainly upon auscultatory percussion for the determination of the gastric area and have verified the accuracy of the method by x rays, outlining prior to operation and necropsy, and other methods. In some few cases, the heart beat against the distended stomach furnishes the percussion, so that we need only move the stethoscope to determine the gastric area. Generally speaking, however, it is better to hold the stethoscope still and to move the percussing finger. I find the ordinary binaural Cammann stethoscope, or rather my simple flexible modification of it, superior to the monaural instrument, and the various forms of the phonendoscope have proved disappointing. In certain cases, as when the colon and stomach are distended and in close contact, it is impossible to discriminate between colon and stomach by ordinary percussion or even by auscultatory percussion. In such cases I use a tuning fork instead of the percussing finger. In my office I often substitute for the percussing finger an electric buzzer with hard rubber stem to communicate a thrill to the stomach or other organ examined. For nice discriminations, however, this is not so good as the slightly more troublesome tuning fork.

In 1897 I attempted to make a radiogram of iron tablets in the stomach, but failed. In July of the same year, through the courtesy of Mr. I. W. Detwiller, an x ray apparatus was placed at my service and a fairly large series of examinations were made with the fluoroscope. Bismuth and capsules containing reduced iron were used to throw a shadow. The results were very satisfactory, but, as in every case, the results of the much simpler method of auscultatory percussion, were verified, I employ the

x rays only in puzzling cases. This examination with the fluoroscope showed why the radiographic method had failed—gastric peristalsis moves the capsule too rapidly to permit a sufficiently long exposure. When bismuth is used free in the stomach, it is at first seen as a dark mass at the bottom, later it becomes distributed over the stomach so as to throw a faint shadow of practically the entire area. In some cases with active peristalsis, the fumbling of the bismuth by the peristaltic wave is beautifully shown. This method was reported in *Medicine*, February, 1898, and Dorn made an independent report in the same month, in a German periodical. My claim of priority has been admitted, in this country, at least.

What may be called combined methods of determining the gastric area depend upon the insertion of some mass into the stomach. In a sense, therefore, succussion of gastric contents and the x ray method just discussed, are combined methods. Among the simpler combined methods may be mentioned distention of the stomach by effervescence, to facilitate percussion, and the auscultation of the sounds produced at the cardia during swallowing. These sounds are conducted with especial loudness only over the gastric area but can, obviously, be continued for but a short time, so that it is hardly practicable to use them for complete mapping out of the area. They are of especial service to determine whether an area of visceral transsonance is really the stomach or something else, as the colon.

External investigation of the gastric area during instrumentation, for the purpose of determining the gastric area, depends upon inspection, auscultation and palpation.

Inspection is applied when the gastrodiaphane is used. This method is not very reliable and is somewhat troublesome, as compared with its small practical utility. The deglutable diaphane, of which Dr.

H. W. Lincoln's instrument is an excellent example, always gives me the same sense of embarrassment as is encountered by a driver whose horse begins to back at a critical moment and who experiences the impossible desire to push on the reins.

Dr. Fenton Benedict Turck used the x rays to determine the position of an opaque instrument in the stomach, before my bismuth method was employed. Indeed, the only points of superiority of the latter are its freedom from objection on the part of the patient and the possibility of seeing the shadow of the whole gastric area at once. Yet, in doubtful cases, the shadow of a bone bulb or of the Turck gyromele is much more distinct.

Fluorescin solutions have been used to intensify the illumination of the diaphane but, in my experience, they have not perceptibly done so. Neither do they afford practical results with the x rays. It has occurred to me to use fluorescent solutions of other kinds to obtain luminous instead of an opaque shadow with the x rays, but quinine and so forth have not proved practicable, the cyanide combinations are toxic, and I have not tried tungstate and analogous preparations, which are not highly toxic. In general it is obvious that an x ray dark shadow will afford a much more definite area than a luminous shadow, however produced; still, the possibilities of the latter are sufficient to warrant a comprehensive attempt at discovery of a practicable means of illuminating the stomach.

Auscultation may be applied to any sound produced within the stomach by any means, such as an endogastric rattle or bell, electric buzzer, etc. In spraying the stomach we may also practise auscultation, and in my method of spraying from the outside end of the tube, a beautiful metallic tinkling may be obtained if the stomach is partly full of water. A whistle may be inserted into the gastric end of the tube, but is scarcely necessary.

Palpation of inserted instruments may be applied to various fairly stiff sounds, with bulbs, but is most practical when Turck's gyromele is used. In the latter case, we palpate the vibrating tissues rather than the instrument.

Internal methods alone may assist in determining the size and position of the stomach, but do not enable a definite area to be mapped out. The distance at which the tube works best affords some vague conception of the altitude of the stomach, and the amount of contents withdrawn may enable the diagnosis of dilatation to be made. However, even in extreme dilatation, the amount of contents does not usually exceed the maximum capacity of the normal stomach, 2,000 to 2,500 c. c., and we must be careful that intestinal contents are not included. Conversely, a small quantity of contents does not exclude dilatation. The old method of determining the capacity of the stomach by introducing water up to its full capacity is dangerous and unscientific. Several years ago I showed that during ordinary lavage the stomach gets rid of 1,000 to 2,000 c. c. of water introduced, probably by passage through the pylorus, even without pyloric insufficiency. This loss may theoretically be ascribed to absorption from the stomach, but all authorities are extremely sceptical as to the possibility of any appreciable degree of gastric absorption.

The altitude of the cardia may be determined approximately, sometimes by merely noting an obstacle to the passage of the tube, fairly well by the use of the sound with bulbous end, very accurately by McCaskey's inflatable bulb, provided we use only gentle traction, or with one of Turck's modified gyromeles.

The practical application of these methods may be illustrated in a less systematic way by the discussion of illustrative cases.

CASE I.—TRANSPOSITION OF VISCERA: Patient (No.

1070 of 1897-'8) presented a resonant area in the ordinary position of the stomach, reaching from just above the costal margin nearly to the umbilical equator, as determined by auscultatory percussion. In the corresponding right area there was found a somewhat smaller and higher transsonant unit. Pulmonary resonance was found lower than usual on the right side and ceased at the sixth left rib. With the stethoscope at various places in the right hypochondriac region, the diaphragmatic line could be made out. No definite area corresponding to the liver could be found on this side. On the left, however, with the stethoscope in the region of flatness, an area was mapped out from the fourth rib to the costal arch, such as is ordinarily found on the right side, except that it was more pointed than usual below; that is to say, instead of crossing the epigastrium horizontally, it followed the angle of the ribs, and their cartilages.

Palpation met with resistance under the left costal arch but not under the right. Auscultation discovered a rather feeble first heart sound in the usual left area but the apex beat could not be felt here. At the corresponding right point, however, the first heart sound was strong and the apex beat palpable. A reversed cardiac area was also demonstrable by auscultatory percussion.

Transposition of the liver and heart was, thus, definitely determined. The splenic area was so small that, in the absence of a palpable mass, its position could not be determined except by inference. However, as the resonant area on the left side was larger than on the right, and really appeared more like a depressed stomach area than that of the left corresponding region, it was perfectly conceivable that the stomach was not transposed but simply depressed by the liver. The appendix could not be determined by palpation and, though the right testicle hung lower than the left, general transposition could not safely be inferred.

The deglutition sounds, however, enabled it to be positively stated that the small area on the right side was the stomach. They were plain over this area,

were heard only very faintly over the epigastrium and not at all in the dubious left area. Thus, the latter must have been distended colon.

CASE II.—HOURGLASS STOMACH: (Simulated by pyloric obstruction and duodenal dilatation.) Patient (No. 21 of 1904-'5) was seen in consultation with Dr. W. W. Jones, of Dayton. The history was that of chronic gastric ulcer with dilatation and pyloric obstruction. The first suspicion of hourglass stomach was derived from the examination by auscultatory percussion, which gave a large gastric area with an incurved greater curvature. Careful, but not leading, questioning elicited the history of frequent vomiting of some drink at the end of a meal, or shortly afterward, without the vomiting of the meal itself. The diagnosis of hourglass stomach immediately suggested itself but, as I told Dr. Jones, the rarity of the condition rendered one slow to make such a diagnosis, and the vomiting could be explained by a dilated œsophagus, or an œsophageal diverticulum. Moreover, such differential vomiting sometimes occurs without any explanation, perhaps on account of a sort of filtering function of the cardia, such as quite regularly occurs at the pylorus. Then, too, an hourglass stomach from the standpoint of physical diagnosis, may be truly such, or there may be merely a partial contraction of the gastric wall, or the hourglass may consist of stomach and dilated duodenum instead of two gastric cavities. An x ray examination agreed with the results of auscultatory percussion, which were repeated on various dates and supplemented with tuning fork examination, always with the same result as to an hourglass area.

At necropsy, the gastric area was found to correspond with that previously mapped out, as well as with the markings made just before section. Symptoms of obstruction had gradually increased up to the time of death and we were not surprised to find that the opening was even smaller than the area of contracture suggested. Indeed, the gastric wall was agglutinated for over an inch, and the hourglass opening would just admit a small lead pencil. Even at necropsy, the diagnosis between true hourglass stomach and pyloric contraction with duodenal dilatation, could

not be made positively on account of poor illumination, the necropsy having been held at night. The necropsy was of no further interest except that the diagnosis of contracted liver, previously made by palpation and auscultatory percussion, as well as by the x ray examination, was confirmed. The decision for or against cancer, which always suggests itself in chronic gastric ulcer, had not been made before death, on account of the negative findings in the stomach contents nor was it possible macroscopically. The pathological examination showed that cancerous disease had developed in the site of the old ulcer and had extended considerably throughout the gastric wall. The lower chamber consisted of dilated duodenum, the pyloric lumen being of the size of a slate pencil. Thus, the differential vomiting remains without explanation but, as is well known, it often occurs without thought of hourglass contraction.

Francis Sedgwick Williams, *Annals of Surgery*, 1900, has collected seventy-five cases of hourglass stomach. In only a few was the diagnosis made before necropsy or operation. Twenty of the cases were apparently congenital, and at least forty-three of the fifty-five others were due to gastric ulcer, although one of the ulcer cases was due to ingestion of hydrochloric acid of corrosive strength. One case was due to cancer originating outside the stomach, and two others to adhesions plainly exogastric in origin. In the remaining nine there were adhesions to neighboring organs and, in some of these, the condition may have arisen outside the stomach. John M. Elder, in Vol. XXXV., 1902, of the same journal, refers to a case operated on under the diagnosis of pyloric obstruction, rectified when the organ was exposed. But he criticizes two other cases treated by gastroenterostomy below the hourglass constriction, so that the operation did no good. Thus it is evident that this condition has an importance to the gastroenterologist and although an exact anatomical and pathological diagnosis may not be possible, it certainly seems that a probable diag-

nosis may be made prior to section, of great practical value to the surgeon and still greater to the patient.

GASTRIC DILATATION.—Although mere enlargement of the gastric area by auscultatory percussion, or as determined otherwise, does not necessarily justify the diagnosis of dilatation, it is of great service in making the diagnosis. We must distinguish sharply among obstructive dilatation, obstruction without dilatation, reflex ischochymia due to pyloric spasm, with or without dilatation, ischochymia due to atony, atonic dilatation, and true gastroptosis. Surgeons have a rule that obstruction at the cardia signifies a small stomach, obstruction at the pylorus, a large dilated stomach. Perhaps this rule ought to hold good, but it certainly cannot be depended on.

CASE III.—For example, a short time ago, I saw just before operation Mrs. T. (No. 1090 of 1904-'5), who was said to have a dilated stomach, for which gastroenterostomy was to be performed. I had previously examined the gastric contents which were practically normal in amount, fluidity, and acidity, though the hydrochloric acid was only 10 per cent. There was no significant excess of lactic acid nor of fermentation acids generally, and neither was there ischochymia though I cannot say but that after other than the test meal, she may have had more or less gastric stagnation. In the few minutes allowed me for examination under anæsthesia, it was made out by auscultatory percussion that the stomach was not greatly dilated, the lower curvature being above the umbilicus. A hard tumor was, however, discovered by palpation of about the size of a small walnut, quite movable and corresponding in range of motion to the pylorus. These results, though scouted by the surgeon, were verified on section. Now although the exact size of the stomach was not of prime practical importance, it is obvious that gastroenterostomy is not indicated unless there is pyloric obstruction and that the operation indicated in such a case is either resection of the tumor as possibly malignant and as a better means of removing such

obstruction as may have existed than gastroenterostomy, or, if resection is impossible and the disease is malignant, the stomach should be short circuited by establishing a superior enterostomy. Gastroenterostomy is not an ideal operation since it establishes an abnormal condition and fails to remove the essential focus of disease. It is, at best, a second choice and should not be undertaken unless more radical operation is contraindicated. In this case, the operation, as prophesied, proved a failure.

GASTROPTOSIS.—This condition is frequently confused with atonic gastric dilatation. Indeed, the latter is a relaxation and sagging of the lower part of the stomach, but the term ptosis ought to be limited to a sagging of an entire organ and this is a practical as well as theoretical distinction because a relaxation of smooth muscle is very different, as to treatment and prognosis, from a relaxation of ligamentous structures. Now it is perfectly obvious that gastroptosis cannot be diagnosed, except by guess, until the whole gastric area is mapped out. Generally this can be done by auscultatory percussion, but in doubtful cases various other measures, such as x ray, must be resorted to.

CASE IV.—Patient (No. 56 of 1899-1900) gave a rather vague history of stomach trouble of eight years' duration. There was no ischochymia but hydrochloric acid was only 2 per cent. while the total acidity was 75 per cent. and there was acetic in excess of lactic acid. The right kidney was movable in the first degree, the left in the second. At first examination, the stomach seemed to be normal in area but we must always remember that auscultatory percussion does not name an area but merely tells us that there is something, of such a form and size, under the body wall and that this something is practically homogeneous in nature. For instance, I have found large hepatic cancers which were perfectly palpable and which, by ordinary percussion gave exactly the same quality of percussion note as the liver, but which were absolutely separable from the liver by auscultatory percussion, whereas on the other hand,

equally large cancers, inclosed in normal liver tissue, appeared as part of the liver. With regard to the gastric area, it must be borne in mind that the colon may displace the stomach or be overlapped by the latter, and in the case under discussion the area which at first seemed to represent the stomach was really the splenic flexure. In this case I attempted to pass the gastrodiaphane which revealed some very interesting conditions. First of all, the cardia was found to be depressed several inches. The diaphane encountered resistance and we could get no luminous shadow of the stomach. The patient vomited about 250 c. c. of œsophageal mucus and, so far as could be judged from the sensation, the kinking of the tube and wires supporting the diaphane, the capacity of the œsophagus as indicated by the mucus, and the failure of diaphany, the diaphane had simply turned on itself in the œsophagus. I may explain that my diaphane consists of an ordinary stomach tube, in the end of which is an annealed glass vial tightly stopped with rubber, through which the wires pass into the little electric lamp which is thus protected against breaking, or rather the stomach is protected from bits of glass in case the lamps should explode. The inflexible end of the apparatus is nearly two inches long. This apparently turned in the œsophagus. More careful auscultatory percussion, while still revealing the same area as originally, now showed the presence of a sling stomach, extending an inch or more above and below the umbilicus and having the characteristic shape of a crooked neck squash. This area was enlarged by blowing air through a stomach tube, while the upper area did not increase in size. Moreover, the air could be heard entering the lower area and not the upper. Even palpation showed that the lower area was in continuity with the œsophagus while, on inflating the bowel through the anus, the upper area increased slightly in size and became more highly tympanitic.

Case V.—Patient (No. 79 of 1903-'4) illustrates the more common type of gastroptosis not amounting to sling stomach but distinct from atonic dilatation. She, by the way, had been much improved in the past by wearing Dr. C. D. Aaron's belt. The lower border of the gastric area, veri-

fied by various means already discussed, came to the umbilical equator. This in itself would not justify the diagnosis of gastroptosis. The majority of such cases should be called atonic dilatation. Here, however, the upper border of the area was at the eighth rib, whereas it should reach the seventh, sixth or even the fifth if the stomach were greatly distended. In such cases, radical cure is practically impossible without gastropexy which is still sub judice, but symptomatic cure can be accomplished by a belt and care as to gastric chemistry and motility.

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DISCUSSION.

DR. ROSE: To discuss this paper fully would take a long time, and I will only ake the opportunity to show a picture of the circumscribing gastrodiaphane of Dr. Kemp, by means of which we can locate the position of the stomach most exactly. There is only one reliable method of locating the position of the stomach, and that is with the gastrodiphane. Introducing the circumscribing diaphane you can carry the light along the walls of the stomach in any desired direction and explore the entire wall of the stomach anteriorly up to the pylorus and along the borders of the ribs under perfect control. Manipulation of the tube of the diaphane now in use, after the electric ball has entered the stomach, frequently causes gagging, and at times vomiting, interfering thus with the accuracy of the method. All this is avoided when the circumscribing gastrodiaphane is employed.

DR. J. KAUFMANN: I usually feel safe in relying on percussion only in finding out the size and location of the stomach. It is a good plan to first determine the area of gastric dullness with the patient in the recumbent position, and then place him on his right side and make out the area of dullness in that position. I regard simple percussion and palpation as accurate as any of the more complicated methods—in fact, more so, and it requires no apparatus.

DR. BENEDICT: I wish to thank Dr. Rose for bringing the circumscribing gatrodiaphane of Kemp to my attention, as it is new to me. In examining these cases, I do not neglect ordinary percussion and palpation, and indeed, the best diagnostician is the one who relies most on his fingers, and the unaided eyes and ears. At the same time, the method I have described with the buzzer is very simple, and does not require much time or apparatus.

[Reprinted from THE MEDICAL NEWS, July 29, 1905.]

A COMPARISON OF METHODS OF LAVAGE WITH THE SIPHON TUBE AND POLITZER BULBS.[1]

BY JOHN P. SAWYER, M.D.,
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IN determining an ectasia or an atony of the stomach our criterion is the test of performance of muscular function as measured by residue of known food found after known periods of time. That this be accurate it is necessary that the amount obtained in the test be as nearly as possible the whole amount at the time present in the stomach. All agree that the recovery of this whole amount is difficult and often not accomplished, especially in the very group of patients in whom it is particularly desirable.

To meet this difficulty by the method of expression commonly used, various modifications have been proposed. Aspirators are numerous, and yet in the popular forms much gain in efficiency can hardly be claimed. Neither Fridlieb's, Boas' nor Straus' exerts more than an interrupted or slight aspiration. The application of a Politzer bag recommended by Ewald, arranged by Kuttner with a hard rubber connection piece, separable for convenience, is the most efficient simple form of aspiration.

The fact that the expression method leaves something to be desired even for the withdrawal of test meals is shown by Fleiner's recommendation, endorsed by Riegel, to have the patient, after expression in the sitting position, lie down for the purpose of obtaining a further quantity likely to be gained after all possible has been expressed by the atonic stomach in the upright body position. It needs but trial in a few cases to convince the investigator that a considerable amount can be withdrawn by the bulbs after all possible has been expressed in the sitting and reclining position.

As examples of the comparative efficiency of expression and aspiration of these bulbs I give a few figures of a trial made on the following plan. They can be multiplied indefinitely but

1 Read before the American Gastro-Enterological Association, New York, April 24 and 25, 1905.

are simply illustrative. In the first trial the patient, thoroughly used to expression and use of the tube, with a considerable motor insufficiency, four hours after the breakfast gave 95 c.c. by expression, and a further quantity of 40 c.c. was obtained by the bulbs. (Expression in the sitting and lying position.) An error of over 30 per cent. is possible by the expression method.

In the second example the patient used to expression and the bulbs, afforded 5 c.c. of thick mucus by the expression in sitting and lying position and 35 c.c. in addition was withdrawn by the bulbs. In this case but 12.5 per cent. of the recoverable stomach contents was obtained by the expression method, and this case also illustrates the advantage of the bulbs in the case of the tenacious mucus of the catarrhal process, in that much shorter tubes afford less opportunity for retarding the flow from the stomach by adhesion of the mucus to the walls of the tube.

The third example is that of a patient considerably used to the tube in whom 60 c.c. was obtained by expression in sitting and lying position, and the use of the bulbs with manipuation gave 20 c.c. additional. In this case 75 per cent. of the stomach contents was recovered by expression.

What is found in practice with the siphon expression in recovering food residue applies also to its use in lavage. In the ordinary sitting position and also in the horizontal position siphoning and expression permit in many cases considerable quantities of water to remain in spite of all reasonable effort. In upright positions the cardiac portion of the stomach often escapes the washing undertaken. Thorough cleansing of the stomach surface is accomplished, if at all, in considerable time and with fatigue to the patient. This thorough cleansing is often not obtained, especially when there is a catarrhal secretion of the membrane. The water goes into the stomach from the tube and back with little motion or current to perform the mixture necessary to establish the cleansing desired. These difficulties are overcome with ease for the patient, thoroughness in the result, speed and satisfaction *to the physician* or nurse, by using the bulbs for

lavage as well as for aspiration of test meals, with manipulation of the stomach, shaking and mixing the solution used with the stomach contents, and later, upon the thoroughly cleansed mucosa.

I use the simple Politzer bulb with the glass connection fitting into bulb and tube. An empty bulb and two or three filled with solutions which are to be employed in succession as desired by the one giving the treatment, are provided. I find it most efficient to have the patient always in the reclining position, clothes thoroughly loosened. The food residue having been aspirated as completely as possible with an empty bulb, a bulbful of solution, medicated as desired, is introduced slowly into the stomach, and free, active manipulation of the anterior abdominal wall over the region of the stomach is used with sufficient energy, sometimes also shaking the posterior surface of the stomach, especially in conditions of atony or ectasia without ptosis. By this is produced a considerable movement of the liquid within the stomach useful for thorough cleansing and bringing the drug in close contact with the cleansed membrane. Then I allow the contents to be withdrawn in the aspirator which has been controlled by pressure with the hand till this time, and cleansing of the viscus can be accomplished more thoroughly, in less time, with less fatigue to the patient, than I can accomplish it by the use of the siphon tube. That the whole quantity used as a wash is withdrawn is seen by the filling of the bulb. The patient is freed from efforts at expression, and lying as quietly relaxed as possible gains much in comfort.

With reference to the thoroughness I would call attention first to the recognized fact that frequently after the water is obtained clear in ordinary lavage there will sometimes be a marked rush of apparently pure food residue which has escaped the ordinary attempt at cleansing. This difficulty I do not find in practice with the procedure described.

As to the comfort of the patient the testimony of those in whom the two methods have been tried is strongly in favor of this method as easier

and more cleanly; and they emphasize the freedom from straining and the annoying gagging or retching sometimes associated with expression.

As to the possible danger of injury, it is subject to the ordinary precautions which we would use in states of active inflammation of the membrane, in conditions of gastric ulcer, perigastritis and the like, which control our application of the siphon tube. Bits of membrane, blood, etc., are not more often found in washing by this method than by expression. My judgment on the basis of five years' routine use is that by the avoidance of straining by the patient the possibility of damage from this cause is lessened.

As to convenience and cleanliness for the operator, my own experience in this respect has been confirmed by many of my colleagues and assistants who using the procedure, express themselves as highly pleased with its convenience, speed and comfort.

I wish to draw especial attention to the advantage in lavage of having the patient reclining with loosened clothes, and then making use of free manipulation of the stomach wall to secure a more thorough cleansing and the more thorough application to the mucous membrane of suitable solution chosen for direct medication. In this fashion all the solutions used may be employed with a reasonable certainty that all portions of the stomach membrane are reached in the treatment and that all of the solutions injected are withdrawn.

[Reprinted from THE MEDICAL NEWS, July 8, 1905.]

CASE OF PEPTIC ULCERS AFTER GASTRO-ENTEROSTOMY CAUSING GASTRO-COLIC AND JEJUNO-COLIC FISTULAE, AND OF SPONTANEOUS CLOSURE OF GASTRO-ENTERO-ANASTOMOSIS.[1]

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THE history of the case which I want to report to you presents a remarkable development of gastric disturbances following a gastro-enterostomy, performed in December, 1901, on a man then forty-four years of age.

During the three years previous to this operation, the patient had suffered periodically from more or less severe gastric pains. Three times severe hematemesis had occurred and once there were all the symptoms of perforation, followed by localized peritonitis in the pyloric region. There was hyperacidity, some motor insufficiency and a pyloric tumor, which during the time of pronounced disturbances could easily be felt as a hard mass and which as a rule became smaller and less distinct after treatment.

Three times a strict treatment for ulcer had been carried out and in addition to this at several periods local treatment, lavage with silver nitrate, bismuth, etc., had been used. Once a long course of specific treatment was given because one of the patient's former physicians had diagnosed syphilis on account of an eruption on the lips and hands which he had treated several years previous to the development of the gastric symptoms and which had yielded only to iodide of potassium. The patient and his former physician denied syphilis most emphatically. However, since there was a difference in the size of the pupils both of which reacted poorly to light, the mercurial treatment was given a trial with no perceptible effect upon the ulcer. Nine months after the specific treatment, the third and most profuse hematemesis took place.

1 Read at the Eighth Annual Meeting of the American Gastro-Enterological Association, New York April 24 and 25, 1905.

After renewed internal treatment for ulcer following this hematemesis had been rigidly carried out for six weeks, the patient vomited great quantities of strongly acid, fermenting masses. mixed with blood, and showed visible gastric peristalsis. These symptoms of increasing pyloric stenosis and the recurrence of hematemesis in spite of all the internal treatment, led to the operation.

At the operation, performed in December, 1901, by Dr. Lange, the pylorus was found to be thickened and its posterior wall adherent. Its anterior surface showed areas of marked thickening and opacity of the serous covering, one spot, probably the seat of the former perforation, being distinctly retracted. Retrócolic posterior gastro-enterostomy was performed. A very high jejunum loop had been taken and since the angle caused by the fixation seemed rather acute, the afferent and efferent portions of the jejunum were connected by an entero-anastomosis. Silk sutures were used. Recovery was smooth, the only disturbance being caused by pronounced constipation during the first week. During the following three months there were no gastric symptoms and the patient gained 27 pounds. After this time gastric disturbances recurred. At first these were of mild character; pressure and discomfort about an hour after meals, but were followed later on (May, 1902) by more severe abdominal pains regularly coming on in the afternoon. On examination, the pyloric resistance was distinctly palpable, the stomach appeared large and atonic, and the stomach contents showed pronounced hyperacidity.

In October, 1902, severe cerebral symptoms appeared; difficulty in speaking and writing, defective memory, muscle tremor, exaggerated reflexes, papillitis on both sides, convulsions and periods of unconsciousness with complete amnesia afterward. Energetic mercury treatment was given and large doses of iodide of potassium for a long period. In February and March a few shorter attacks of mental disturbance occurred. After this there were no more cerebral symptoms.

In April, 1903, the abdominal pain which had

hitherto been of a moderate character, became very severe. It generally came on in the afternoon or at night and was of such severe character that nothing but morphine would relieve it.

As a rule this pain was located in the upper abdomen, but at times it irradiated into the lower. While it lasted, the transverse colon could often be felt as a stiff, sausage-like tumor, which became softer when the pain grew less. This feature was especially noticeable after the administration of morphine. One could often feel gas escape with the relaxation of the tumor.

For periods of weeks and months the pain came on almost daily. There were other periods of weeks and even of months of perfect freedom from abdominal pain, especially in the summer of 1903 and 1904, while the patient was in the mountains. In November, 1903, six months after the abdominal pain first became severe, the patient complained for the first time of sulphurous taste and belching for several days and then vomited large quantities of fluid fecal matter. Lavage of the stomach was begun. The vomiting was not repeated but the complaint of fecal taste and the finding of fecal matter in the stomach became more and more frequent and finally, during the winter of 1904-1905, became constant. At first there were periods of weeks and months when stomach lavage performed at any hour of the day showed absolutely no fecal matter. During such periods, which were noted as late as the summer of 1904, examination of the fasting stomach and after test-meals showed pronounced hyperacidity and good motor activity, or at most, motor insufficiency of a mild degree. Even at the periods when fecal matter was found in the stomach, there were no signs of stagnation of food taken by mouth.

At the periods when fecal matter got into the stomach, it was often found in the fasting organ. Very often it entered the stomach only after a meal. The patient could recognize it by the fecal belching and could verify it by removing fecal matter from his stomach by the tube, even when the stomach had been cleaned by lavage before the meal. The same phenomenon was observed during lavage. After the wash-water

had returned from the stomach perfectly clear several times, it would suddenly become mixed with fecal matter. Sometimes the flow of fecal matter would continue as long as the washing was kept up. The quantities of fecal matter thus removed by the tube were often very large.

Occasionally small formed feces were found. As a rule, however, the fecal masses were more fluid; they were in every way like the fecal matter obtained by defecation, in color and smell, in macroscopic and microscopic appearance. When removed from the stomach in the morning or a number of hours after meals, they never showed undigested food remnants.

The feces obtained from the rectum were large, semisolid, mostly light-gray, often acid from gas fermentation, showed enormous quantities of bacteria and were always well digested. Undigested food remains were never found, even when the stools were washed through a sieve. The chemical examination showed neither hydrochloric acid nor pepsin; albumin digestion examined by Mette's method, proved negative. Defecation was on the whole regular but at times, especially during attacks of severe pains, constipated.

There was no stagnation in the stomach of food taken by mouth; fecal matter would enter the stomach, but we could not demonstrate that stomach contents entered the colon. The existence of a gastro-colic fistula was finally proved by the following test:

A prolonged lavage of the stomach with consequent removal of great quantities of fecal matter was directly followed by a prolonged lavage of the rectum and colon. When the wash-water from the colon appeared somewhat clear, a suspension of lycopodium stained by gentian violet was introduced into the colon under slight pressure. A second lavage of the stomach carried out immediately afterward again produced great quantities of fecal matter, which on microscopical examination proved to be thoroughly mixed with the stained lycopodium.

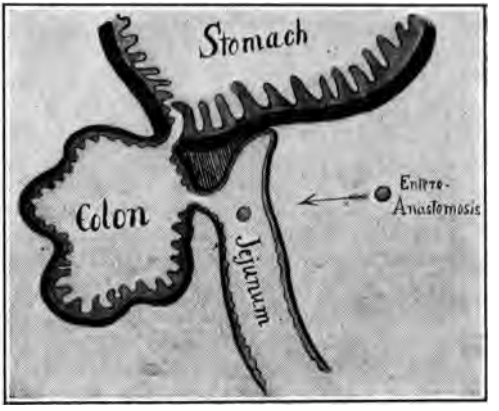
During the winter of 1904-1905 the condition *became unbearable*. The severe abdominal pain

occurring at all hours of the day and at night made the patient a confirmed morphinist. Three more courses of specific treatment (April, 1903, June, 1903, January, 1905), had no effect; nor did any other treatment give relief.

The patient, being very much annoyed by the constant flow of fecal matter into the stomach readily consented to the proposed operation.

The operation performed by Dr. Gerster (March 7, 1905), showed the middle portion of the transverse colon to be somewhat stiff and narrowed. It was adherent above to the larger

Fig. 1.*



curvature of the stomach, and posteriorly to that loop of the jejunum which had been used in performing the gastro-enterostomy. The apex of this greatly dilated loop was hidden behind the colon. After turning up the colon, we found a connection between the posterior wall of the colon and a diverticulum of the jejunum, which must have been formed by traction. On section, this connection proved to be an open channel of considerable diameter between the cavities of the colon and the jejunum. The edges of the openings were soft and covered by mucous membrane. They showed neither infiltration nor ulceration. Both openings were closed by a Lembert suture. Then the colon was reflected and after the removal of a number of omental adhesions, it became evident that there existed another and

larger connection between the upper surface of the transverse colon and the posterior wall of the stomach, which on dissection was also found to be an open channel uniting the cavities of the stomach and the colon. These two apertures, the edges of which were also soft and covered by mucous membrane, were sutured. It then became evident that the two openings connecting the colon, on one side with the stomach and on the other with the jejunum, were quite near together. The colon, which was already somewhat narrowed, became still narrower in consequence of this double suturing, to such an extent as to cause fear of a pronounced stenosis.

We had the choice of resecting this narrowed portion of the colon or of connecting the ascending colon with the sigmoid flexure. The latter proceeding was adopted because the intimate and extensive adhesions of the colon to the posterior parieties made resection a very hazardous undertaking. Since the operation had already lasted three hours, the Murphy button was used.

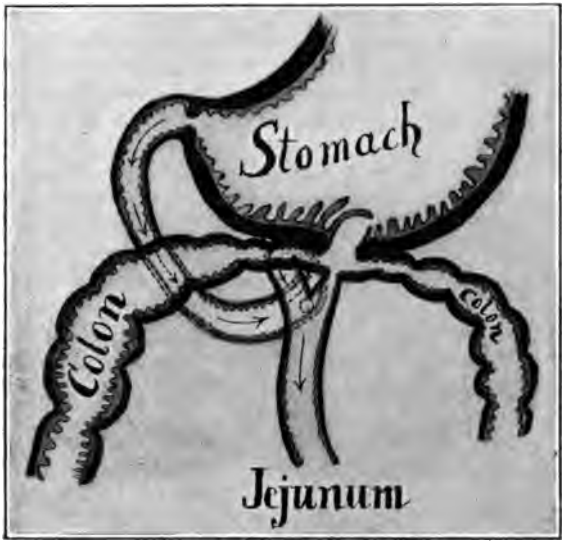
Recovery seemed uninterrupted during the first six days after the operation. Suddenly, on the sixth day, symptoms of perforation developed, followed by collapse. Immediate laparotomy showed that the perforation was caused by a continued necrosis of the sigmoid flexure, issuing from the site of the Murphy button.

I quote a few points from the post mortem: "On studying the relation of the fistulæ after removing the sutures, we found: (1) A hole in the stomach, surrounded by enormously hypertrophic folds of the gastric mucous membrane, one of which covered the hole from the gastric side completely and acted as a valve. (2) That the two holes in the colon were quite close to each other. In cutting the sutures the intervening bridge was destroyed. The upper hole corresponded to the hole in the stomach, and the lower one to the opening in the jejunum. The gastro-colic and the jejuno-colic fistulæ were opposite each other, so that a glass probe could be passed directly from the stomach through the two holes in the colon into the jejunum; (3) That the hole in the jejunum was situated in the *diverticulum* of the efferent portion, which was

very much dilated; the afferent portion was short and formed a straight line from the plica duodeno-jejunalis toward the stomach.

Absolutely no traces could be found of the opening between the jejunum loop and the stomach, which was made at the primary gastro-enterostomy. The apex of the jejunum loop was adherent to the lower surface of the transverse mesocolon, but it was entirely separated from the stomach. The opening of the entero-

Fig. 2.*



enterostomy, though small, was still patent; it was in close vicinity to the jejuno-colic fistula, which was about 5 cm. below the apex of the jejunum loop. The pylorus was patent, but at one point markedly narrowed, showing on its inner surface the scar of the old ulcer.

The transverse colon at the level of the fistulæ was thickened and measured only 7 cm. in circumference, while the hepatic part of the transverse colon was dilated to 10 cm. circumference and thinned out. The descending colon measured only 4¼ cm.

* The drawings do not give the exact anatomical relations and are meant to be only diagrammatic. The jejunum-loop is here shown attached to the stomach, in order to demonstrate the situation of the former gastro-entero-anastomosis.

There were no anatomical evidences of syphilis, either in the vessels or the viscera (including the brain and the testicles). The larger branches of the abdominal aorta showed slight arteriosclerotic changes. The smaller ones and the thoracic aorta and its branches were free.

In reviewing this case, we must consider separately: (1) The closing of the gastro-entero-anastomosis. (2) The formation of the fistulæ.

The gastro-entero-anastomosis had entirely disappeared. The closing of the anastomotic opening was so complete that it was impossible to locate it. Even the connection between the jejunum-loop and the stomach did not exist any more; the jejunum loop was entirely separated from the stomach. We cannot say how soon after the operation the anastomosis had closed up. So far only a few anatomical facts have been reported, which give information about the final outcome of gastro-entero-anastomosis in benign cases.

G. Gayet described a case which showed the same conditions as our case. He found at a second operation performed one year after gastro-enterostomy, that the first opening had completely closed, that the intestine had been absolutely separated from the stomach, and was only kept adherent to the transverse mesacolon.

Mayo, Feldmann, Robson and Moynihan have reported cases in which a second operation was performed, because the anastomosis had closed. In most of these cases it is simply stated that the opening had contracted; in only one case is it specifically stated by Robson and Moynihan, that the opening had completely closed. There are further a few reports of findings at post-mortems: Ferrari found in one case, three years after operation, an anastomosis of only 2 mm. diameter; in another case, six months after operation, an opening of 4 mm. diameter. Jaboulay reports an autopsy after six months, another after a year, with the opening very much restricted. In another group of cases, the opening was found still patent; in Cordier's, even six years after the operation. I saw the opening patent in a case (not published); two years after *the operation.* More facts must be obtained be-

fore we can explain why the anastomosis remains open in one case and closes in another. The complete disappearance of all traces of the anastomosis in our case is certainly very remarkable.

In regard to the development of the fistulæ I would like to suggest the following explanation: The abdominal pains from which our patient suffered so severely must be attributed to a new gastric ulcer which developed near the former gastro-entero-anastomosis. We may class this ulcer with the group of peptic ulcers following gastro-enterostomy. Tiegel has recently published a review of 22 such cases, in which the peptic ulcer was demonstrated either at the post mortem or at a second or third operation. In most of them the peptic ulcer was found in the jejunum. In some it developed directly on the edges of the gastro-entero-anastomosis. In one case, reported from Czerny's Klinik, a peptic ulcer had developed in the stomach near the gastro-entero-anastomosis, caused adhesions to the colon, and had then perforated into the colon, thus forming a gastro-colic fistula.

The same process, in all probability, took place in our case, but in addition, after the gastro-colic fistula had developed, a second peptic ulcer formed in the colon, opposite the fistula. This second ulcer in turn led to adhesions to the jejunum, into which it perforated, thus forming the jejuno-colic fistula. As a result of these ulcerative and perforating processes adhesions were formed between the stomach and colon, and the colon and jejunum leading to a narrowing of the involved part of the transverse colon.

During life this section of the colon could often be felt as a sausage-like tumor, stiffened by spastic contractions. The spasms were probably caused by the irritating effect of the highly acid gastric secretion on the colon, in the same way in which pylorospasm is caused in case of pyloric ulcer. The frequent spastic contractions of the colon may have contributed to another effect of the adhesive processes, *i.e.*, the formation of the diverticulum of the jejunum by traction.

The fact that we did not find undigested food in the feces is fully explained by the presence of

the large fold of the gastric mucosa which, acting as a valve, easily prevented the passage of formed masses from the stomach into the colon, although it could not prevent the flow of gastric secretion into the colon.

The manner in which these peptic ulcers develop after gastro-entereostomy is not yet clear. According to Tiegel, it does not seem that the technic of special methods has great influence, since a study of his 22 cases shows that they occur after all the different methods of operating. Since they occur only in cases of benign diseases of the stomach, it is probable that on the whole the conditions for the development of such peptic ulcers are the same as those for the development of the primary ulcer, *i.e.*, chronic gastritis, hyperacidity, circulatory disturbances, etc. The locality, however, in which the peptic ulcer appears, makes it certain that the operation itself is the direct cause of its development. Perhaps lesions of the mucous membrane, created at the operation, form the basis for the peptic ulcer. Tiegel names arteriosclerosis as a contributory cause. It hardly touches the question in our case, since the arteriosclerotic changes were only moderate and confined to the larger branches of the abdominal vessels.

We have further to consider the question of syphilis in our case. Even if we assume that the patient at one time had syphilis, there is nothing that would permit us to connect it with the formation of the gastric ulcers and the fistulæ. A thorough anatomical examination of the abdominal viscera showed no signs of syphilitic processes, nor did the repeated and prolonged anti-syphilitic treatment yield any apparent result.

At present, syphilis is frequently accused of causing gastric ulcer. However, in only a comparatively small number of cases, has this connection been proved by anatomical examination (Flexner-Trinkler). The mere fact that a patient with gastric ulcer suffers or has previously suffered from syphilis cannot be taken as sufficient proof. In our case the thought of syphilis *was for a long* time misleading in the interpreta-

tion of the predominant symptoms. The attacks of severe abdominal pain, which occurred after the development of cerebral symptoms, we at first regarded as gastric or enteric crises caused by syphilitic disturbances of the cerebrospinal system. Even when fecal matter was found in the stomach, it was considered doubtful whether we had to deal with organic or only functional disturbances, because the fecal matter was found only during certain periods, while at other periods of weeks and months the stomach was free, and also because the patient showed other symptoms of hysteria. The lycopodium test settled this question definitely, proving the presence of a gastro-colic fistula.

It is probable that a knowledge of the frequency of peptic ulcer after gastro-enterostomy would have made clear the true nature of the disturbances in our case before the post mortem.

The development of peptic ulcer after gastro-enterostomy is by no means rare. The 22 cases collected by Tiegel are only cases in which the peptic ulcer was demonstrated at a second or third operation, or at the post mortem. Eight of these 22 cases had shown absolutely no symptoms until suddenly perforation occurred. We may, therefore, conclude that in all probability a great number of cases escape detection.

We ought to bear in mind the possibility of peptic ulcer, whenever gastric disturbances recur after gastro-enterostomy. In fact, we should keep it in mind whenever gastro-enterostomy has been performed and regulate accordingly the treatment after the operation. The great immediate relief after such operations and the apparent immunity with which ordinary meals are digested, too often leads the surgeon to allow a liberal diet. It should be remembered that the chronic gastritis and the hyperacidity which accompany the primary ulcer, do not disappear immediately after operation, and that if not treated properly they may contribute to the development of new ulcers. The further surgical treatment of peptic ulcer following gastro-enterostomy has given very poor results. Complete recovery is rare; fatal issues frequent.

A careful consideration of all these facts

should tend to restrict the indication for gastro-enterostomy, which otherwise gives such excellent results in certain benign gastric diseases.

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DISCUSSION.

DR. A. G. GERSTER, of New York City: When a surgeon faces a problem such as has been presented to us in this case, there is involved in the solution of it a considerable element of uncertainty and hazard, which makes the undertaking all the more interesting. The very painstaking, exact and laborious tests made by Dr. Kaufmann had in a general way given us the facts in the case; namely, that a communication existed between the stomach and the colon, but of the second communication we had no idea.

When the abdominal cavity is opened by the surgeon, conditions may confront him that are in no wise explicable except by an autopsy investigation of the relations of the organs involved, and upon these topographical facts, which no previous examination can throw light upon, depends whether the surgeon will be able to afford temporary or complete relief. This injects into the surgical treatment an element of uncertainty.

In this case the chief difficulty was encountered in ascertaining the actual condition of affairs. We know that in dealing with these conditions we are apt to find extensive adhesions between the various organs, and to clear up the field so as to get a correct comprehension of the existing state of affairs is sometimes the most difficult part of the operation; at least, that has been my experience. In this particular case, it took us over three hours to clear up the field. The dissections had to be made very cautiously, so as not to injure the intestines, and on account of the communication between the stomach and the colon, it was never safe to assume that faecal matter was not present in either or both of these organs, in spite of thorough lavage.

Finally, we succeeded in clearing up the field, and having first sutured the communication between the colon and jejunum, which was comparatively simple, we were ready to deal with the communication between the colon and the stomach. Both of these fistulous openings were very close together, which fact added to the difficulties of the operation. After closure of the gastro-colic fistula, the patient's condition was still fair, in spite of the fact that he had long suffered from mal-nutrition. He was an old morphine habitue, and these, as I know from experience, mostly bear operations excellently. As the lumen of the transverse colon had been very much reduced by its double suture, it was deemed necessary to establish a communication between the ascending colon and sigmoid flexure by means of a Murphy button. After the insertion of such a button, marginal necrosis always occurs. It is necessary in order to permit the exit of the button; but in this case the necrosis extended far beyond the ordinary limits, and led to perforation and to the death of the patient six days after the operation. I believe the prognosis would otherwise have been good, as the gastric, colic and jejunal sutures all were found sound and practically healed at autopsy.

I have since thought that in establishing a communica-

tion between the colon and the sigmoid flexure, the use of sutures would have been preferable to the button, but on looking up the literature on the subject I have found that necrosis has followed the use of sutures as often as that of the button.

I believe the explanation offered by Dr. Kaufmann as to the cause of the fistulae in this case is very plausible and interesting.

Before concluding, I wish to use this case as a text, and say a few words to those who are so ready to blame the surgeon when he is obliged to do his work under most hazardous conditions, and when so many factors, such as the anaesthesia , the respiration, the general condition of the patient, etc., have to be taken into consideration. In some instances, even on the post-mortem table, where the factor of time does not enter into consideration, the skilled pathologist has the greatest difficulty to establish the exact and true condition of affairs, and how much more difficult, then, is the task of the surgeon?

In the case under discussion, the various fistulae were the result of a peptic ulceration. The recognition of this condition, and its true appreciation, will materilly influence the future therapy of gastric ulcer and pyloric stenosis, for the relief of which surgery is so often invoked.

DR. S. J. MELTZER: I would like to emphasize a part of the history of the case reported by Dr. Kaufmann. This patient was under treatment for neurasthenia, and the case for a long time was regarded as one of hysterical anti-peristalsis. This diagnosis was based on the fact that the man actually had a severe intercurrent nervous affection.

There are many cases of ileus that are considered as wholly or partially hysterical, but the possibility of a real anatomical lesion, such as existed in this instance, should always be kept in mind in dealing with this class of patents.

DR. KAUFMANN: The erroneous diagnosis of hysterical anti-peristalsis, to which Dr. Meltzer referred, was induced by two factors: first, the fact that the man gave a history of syphilis; and second, the nervous symptoms that he developed. There was a dfference in the size of the pupils, and very pronounced cerebral symptoms, pointing for a time to paresis or gummatous meningitis. The abdominal symptoms coming on after the cerebral symptoms, with intervals of perfect freedom from pain, were at first taken for attacks of gastric crises. The existence of a peptic ulcer was not suspected. The abdominal symptoms were regarded in the nature of a functional disturbance, particularly as they were so intermittent in character. The finding of faecal matter in the stomach was very irregular, and there were intervals of months when no faecal matter could be found in the stomach.

The important lesson to learn from this case is the possibility of the occurrence of a peptic ulcer after gastroenterostomy.

ETIOLOGY AND SERUM TREATMENT OF DYSENTARY: Discussion opened by Dr. William H. Park, of New York City. This subject has been written about so much during the past eight years, that it is difficult to select points to discuss. As to the bacteriology we might first touch on the proof that bacilli are the exciting factor in dysentery. So far as my experience goes, and it is in accord with that of others, amoebic dysentery in the United States is a rare disease. Most of the examples met with are chronic cases that have been infected in semi-tropical or tropical climates.

Among the half a dozen cases of chronic dysentery that I have examined, three were infected with amoebae, two with a certain type of colon bacillus; one with the para-dysentery bacilli. In none of them was the bacillus of Shiga found.

From the results of examinations in hundreds of cases of acute dysentery, I am certain that in characteristic cases of dysentery—that is those having tenesmus and bloody stools—we can just as surely expect to find bacilli of the group which we call the dysenteric group as we can expect to find diphtheria bacilli in diphtheria, providing, of course, correct technic is used. These bacilli belong to a group that has given rise to more or less confusion. When Shiga, in the Japanese epidemic of dysentery, found a definite and before unrecognized bacillus in the mucus that came from the inflamed lower portion of the intestines, and after death found it in the mucous membrane, but not in the blood, and also found that after a few days specific agglutinins were present in the blood, he came to the conclusion that the bacillus now called the Shiga bacillus, was the exciting factor in all cases of dysentery not due to the amoeba. He did not work out carefully, however, all the characteristics of the bacillus, and a little later Kruse, Flexner and others isolated bacilli in other countries which agreed with the published characteristics of the Shiga bacillus. They naturally came to the conclusion that they were all dealing with a single variety of bacillus. Subsequently, however, investigators in Germany and in this country comparing the cultures found that these bacilli were not all the same. The Shiga bacillus, was indeed found in Japan, Manila, America and elsewhere, but there were other types of bacilli, which were found usually in mild, but sometimes in severe cases. These bacilli had different agglutinating properties, and were dissimilar in other respects. These other bacilli were found not only in cases of dysentery, but also in cases of summer diarrhoea.

Even with our present knowledge, I think we can say definitely that in the more severe epidemics, throughout the world, the Shiga bacilli will usually be found. They are abundantly present in the mucus during the heighth of the disease; their number diminishes with recovery and finally disappears in convalescence. In addition to these severe outbreaks of dysentery, there occur mild epidemics scattered throughout the world. These are excited by a

number of varieties of bacilli, which can best be designated as para-dysentery bacilli. These differ among themselves, and represent perhaps half a dozen or more different strains. The great majority however belong to two varieties. It has also been found that the agglutinating and bactericidal serum produced by any one of these dysentery and paradysentery bacilli, although acting most effectively on its own strain, will act to a certain degree on all the strains.

Are cases of so-called summer diarrhoea also due to the Shiga bacillus? I think that all of us who have been interested in investigating this subject are now agreed that the Shiga bacillus is very rarely found, excepting in cases of true epidemic dysentery. On the other hand, it is equally certain that the so-called para-dysenteric bacilli are met with in quite a number of cases of diarrhoea—mild and moderate cases of so-called summer diarrhoea, that present no definite symptoms of dysentery. In a great many of these cases, however we cannot find, after a most careful search, any of these paradysentery bacilli, nor can we find them in so-called cholera infantum. As these paradysentery bacilli are found in a small percentage of normal cases, their occasional occurrences in summer diarrhoea without dysenteric symptoms does not indicate that they are an active factor in exciting this disease.

The serum treatment of dysentery. I think many of us are beginning to feel that a further increase of knowledge concerning bactericidal sera may, instead of teaching us how to cure, teach us why we cannot expect any great results from serum in many of the bacterial infections. My experience with serum therapy in dysentery is limited to the observation of about thirty cases. Thus far the anti-dysenteric serum placed on the market is not standardized, so the dose must be measured by the cubic centimeter.

According to the reports that have been received from cases of true epidemic dysentery, treated with the anti-dysenteric serum, in about two-thirds of the cases the symptoms of the disease were apparently ameliorated by the remedy, while in the remaining one-third it seems to have had no effect. Shiga, and others who have had the most experience with it, have great faith in its efficacy. From my own experience, I believe that when you have to deal with a severe case of true dysentery, it is well worth while to begin injections at the earliest possible moment and give it in doses of 20 c. c., t.i.d., for four or five days, or until the symptoms subside. If the injections are stopped too soon a relapse frequently occurs. When we come to ordinary summer diarrhoea, however, I have never seen any good whatever result from the use of the serum. In these types of the disease the paradysentery bacilli are apt to be the type present and they are usually only a part of a mixed infection being associated with colon bacilli or other bacteria. In these cases the serum is of no benefit, so far as my experience goes. A great many of the cases of acute dysentery in this climate

are so mild that if the physician sees them early and prescribes proper medication and diet, recovery promptly takes place and serum treatment is unnecessary. The use of the serum seems indicatd, therfore in the rather limited number of cases in which the symptoms are both severe and characteristic of dysentery.

DR. JOHN H. HUDDLESTON, of New York City: I have had several cases which were of the type described by Dr. Park as para-dysentery, which ran the usual course, lasting about a week or ten days. In not a single one of these cases could we discover the origin of the disease. The patients were not run down, but, on the contrary, their previous condition was distinctively good.

Although this disease is usually considered as one of warm weather, the worst cases I have seen occurred in the middle of winter. Of the rather limited series that I have observed, one-fifth occurred between November 1st and April 1st of the following year. All the patients recovered. Serum was not used in anv of them. The convalescence was somewhat prolonged and, while the bacilli disappeared promptly, the abnormal condition of the stools persisted for a long time. Sometimes they were normal for three or four months; then for several days they contained mucus, but no bacilli. The attacks lasted the usual length of time, seven, ten to fourteen days, and the passages were most frequent during the first stage of the disease, and gradually diminished in number. There was usually considerable abdominal pain, and invariably sensitiveness over the ascending or decending colon. The treatment consisted of saline irrigations, with salol and castor oil internally, and morphine when necessary.

[Reprinted from THE MEDICAL NEWS, July 22, 1905.]

CLINICAL FEATURES OF THE BENIGN STENOSES OF THE PYLORUS.[1]

BY HENRY L. ELSNER, M.D.,
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THE consideration of the Clinical Features of the Benign Stenoses of the Pylorus with their medical treatment, the subject which your presiding officer has invited me to consider in this symposium, would indeed seem to be hackneyed and threadbare were it not for its growing importance from a therapeutic standpoint. It may be said that we are passing through a transition period so far as the treatment of these conditions is concerned. Interesting experiences with individual cases, often unique, with the association of surprising pathologic finds which have finally cleared the horizon, still further justify the discussion which you have arranged for this meeting. The writer, in accepting your invitation, assures you that he fully appreciates the included privilege and honor. He will consider in his treatment of the subject those abnormalities, benign in character, within or without the stomach, which block its outlet sufficiently to interfere in any degree with the emptying of that organ or otherwise disturb its functions.

The benign stenoses of the stomach may be either (1) *functional,* or (2) *organic.* The latter may be either (*a*) congenital, (*b*) acquired. The acquired stenoses may be either intrapyloric or extrapyloric.

1. The *functional stenoses* are likely to be transitory, the patients highly neurotic. We have occasionally assumed the presence of *pyloric spasm* in cases where gastrectasia had persisted for months without producing symptoms, in which there followed periods during which the stomach became intolerant; there was peristaltic

1 Read before the American Gastro-Enterological Association, in New York, April 24 and 25, 1905.

unrest with hyperacidity. In some the symptoms were aggravated by mental distress or other causes, including mainly those which ungeared the nervous system. Neurasthenia was always in the ascendency. In none of these cases did the evidences of stenosis become permanent, in none was there reason to suspect grave pathologic conditions.

There are also cases of *intermittent hyperchlorhydria without ulcer,* in which we assume the presence of pyloric spasm recurring at intervals of several weeks or months. There are severe pains, intolerant stomach, nausea, vomiting of large quantities of hyperacid fluid, severe headaches and often cerebral symptoms. With the epigastric pain, which may become severe, there is eructation of gas, visible peristalsis, constipation, a great thirst, complete anorexia. The total acidity is abnormally high so long as the symptoms persist, due to the presence of HCl. Young adults are most likely to develop these symptoms, and errors of diet, excessive use of sweets and mental emotion are leading factors. The chemical examination of the stomach contents differentiates these cases of probable spasm of the pylorus from ordinary migraine and gastric catarrh.

Occasionally we find a case of *acute dilatation* of the stomach with alarming symptoms, collapse without previous history of stenosis or other stomach disorder sufficient to direct attention to that organ. Some writers have reported deaths during such acute dilatation. Whether these cases are to be classified under pyloric spasm, as was suggested by Pepper and Stengel, has never been definitely decided. Robson and Moynihan speak of neuroparesis in connection with such serious, suddenly arising conditions. A few of these cases are recorded, promptly following operations in which it may be assumed that adhesions or other complications were more likely to have been the pathogenic factors than spasm or so-called neuroparesis. Two cases of acute dilatation seen by the writer seemed, in spite of the fact that there was no proof to justify the strong suspicion, due to overpowering toxemia.

The true pathology of these serious and sometimes fatal cases can be decided only by the anatomic study of the individual case, when in all probability we shall find more than functional disorder.

Pyloric spasm may safely be considered secondary in the majority of cases and its presence must always lead to a strong suspicion of associated organic disease. On several occasions patients beyond middle life have been seen in consultation with symptoms of such spasm recurring at varying intervals, at times there was normal but gradually reducing HCl. production; in other cases the free HCl. was slightly reduced; in all of these lactic acid was absent. The end proved that we were dealing, at the time of the supposed spasm, with incipient malignancy. The urgent need of repeated chemical tests and physical examination at short intervals without undue haste to reach conclusions, must be emphasized in these cases.

The question has often arisen in my mind whether pyloric spasm is not occasionally present in neurotic girls, particularly during the menstrual period, with intolerant stomachs, marked and painful cramping of the organ, visible and persistent peristalsis. It is only exceptional that these hysterical girls have failed to vomit. The pain with peristalsis has persisted, yielding finally, not to alkalies but to appreciable doses of belladonna. The associated dilatation of the stomach has not persisted beyond the acute period.

It will be wise to hesitate long before diagnosticating a primary pyloric spasm in the adult, for it will be necessary to exclude organic disease by a thorough consideration of the separate functions of the stomach and the final elimination of mechanical organic obstruction at the pylorus.

Let us leave this part of our subject with the positive assurance that as we become more scientific we are the more thoroughly convinced of the rarity of functional disease and that, in considering the clinical features of pyloric stenosis, supposed to be dependent upon transitory nervous influences, we assume a mental attitude is

which there is a fixed determination to accept such diagnosis, only after the greatest possible justified resistance.

2. *Organic Stenosis.*—A. *Congenital stenosis* of the pylorus may be of moderate degree, in which patients live for years with more or less continuous symptoms, a second class of cases in which the symptoms during the first year or two of life are sufficient to justify a suspicion of marked pyloric stenosis, in which, however, nature comes to the rescue, occasionally supplying such infants with almost normal functionating stomachs during life, and finally that serious class of cases in which symptoms are likely to follow birth, as a rule before the fourteenth day of life, never postponed beyond the ninth week (and this only rarely) in which death follows before the fourth month unless these infants are relieved by surgical interference.

It is not within my province at this time to consider the theories of the production of these stenoses known as spastogenic and hypertrophic, nor the Freund hypothesis which practically includes both spasm and hypertrophy as the cause of these. The writer recalls cases of congenital stenosis of moderate degree, in which all symptoms justified the diagnosis, which have yielded to proper treatment. Compensatory hypertrophy sufficient to overcome moderate mechanical obstruction must have followed. These cases include the child of a colleague in my own city with symptoms beginning at birth in which the child during the first two years of his life presented all of the symptoms of congenital stenosis, in which the boy, to-day twelve years old, is to all appearances perfectly well. In speaking to the father of the child while writing this paper, I was reminded of the fact that when the boy was one year old he weighed less than he did at birth, that his stomach was at that time still monstrously dilated, holding stagnant food, and that there was persistent peristaltic unrest.

The symptoms in all cases of congenital stenosis must follow birth immediately or cannot be long postponed. In four cases seen by the *writer*, three confirmed by post mortem, vomit-

ing and other symptoms of indigestion first showed themselves, in the first before the end of the second day, in the second and third during the second week and in the fourth during the fourth week. In all of these cases there was marked visible peristalsis. It was by no means easy to detect the abnormally resistant pylorus; in all, the stomachs were greatly dilated, constipation and vomiting were constantly present.

Persistent vomiting in children, beginning after the ninth or tenth week of life, is not due to congenital narrowing of the pylorus. The majority of children with the congenital defect die before the end of the third or fourth month. Persistent vomiting in babes which begins shortly after birth is not as a rule to be interpreted as dependent upon congenital stenosis. Such conclusions would lead to great harm. Forcible projectile vomiting is quite characteristic of stenosis. The food seems to be held, only that it may be thrown far from the mouth and this suddenly, as a rule without warning. This is as true of breast-fed children as of those artificially and properly nourished. *No possible modification of the food relieves the symptoms where the stenosis is of high degree.* Single feedings may be held for a time and vomited with a second or third nursing. Couple these characteristics with marked visible peristalsis, even without palpable thickening, occasionally a high-pitched percussion note corresponding with the location of the pylorus, the physical signs of stomach dilatation, and the diagnosis becomes reasonably certain. Rapid wasting with many of the other symptoms found in the adult, as a rule ends the scene. *Congenital incomplete stenoses of the pylorus are frequently the cause of chronic disturbances,* as already mentioned, *which continue throughout life,* including marked and persistent gastrectasia, motor insufficiency, anomalies of secretion, symptoms of Leube's "nervous dyspepsia," including marked neurasthenia. These cases present a continuous train of symptoms with periods of marked exacerbation. While writing this paper such a case was sent to me in which a man forty-three years

of age had never been without symptoms of stenosis and gastrectasia. The stomach is never free from stagnant food. His attendants have never failed to speak of his monstrous and persistent dilatation of the stomach, the palpable pylorus has been a surprise to all. This case is associated with hypersecretion and hyperacidity, peristaltic unrest, an enormously dilated, horseshoe shaped stomach. There is no history of ulcer. In spite of the relief of subjective symptoms by well-regulated treatment, the physical signs persist. Final gastric fibrosis, as described by Hanot and other French and German observers or the hypertrophic pylorus of Cruveilhier and Andral are sequelæ which materially influence the size and position of the organ as well as its functions in cases with almost parallel histories.

B. *Acquired Stenosis.—Intra-pyloric.* (1) *Cicatricial stenosis* (*chronic*). The majority of benign stenoses are unquestionably due to ulcerative processes invading the pylorus. It is not always easy to get a clear history of preceding ulcer. That occasionally ulcers have existed without giving rise to symptoms there can be no doubt. As a rule, however, the previous history is direct, and close questioning with a thorough consideration of all that the case presents, proves the likelihood of previous destructive disease which justifies the reference of stenotic symptoms to consecutive cicatricial changes. If all cases of ulcer were associated with hemorrhage, chronic cicatricial stenosis due to this condition might be readily explained, for there is no symptom which the average patient remembers longer than hemorrhage, particularly when such alarming symptom is associated with severe pain. In these cases we are not limited to subjective symptoms alone or to the consideration of the previous history which may be vague and misleading. The physical examination of the patient, the absence of a large mass, the presence of dilatation, the revelations made positive by the stomach tube and an examination of the stomach contents, will be likely to clear the diagnosis. In those cases of chronic cicatricial stenoses

which follow corrosive poisoning, localized phlegmonous inflammations and other chronic destructive changes, the previous history with a consideration of the clinical features and the absence of a large palpable mass will aid materially in confirming the diagnosis.

The writer feels justified in making the positive statement that in rare cases (and he has met with two of these in practice), there may be cicatricial stenosis of the pylorus following ulcer which has not undergone cancerous degeneration in which cancer nodules have been present in other organs of the body. The last case of this kind which came to his notice was that of a woman who had carcinoma of the uterus and liver with a benign cicatricial stricture of the pylorus. The persistent dilatation and evidences of benign stenosis covered a period of many years, following all of the classical symptoms of ulcer of the stomach.

(2) *Fibrotic stenosis.* (*a*) *General* and (*b*) *localized.* (*a*) *General gastric fibrosis* described by Brinton and Hanot may lead to far-reaching change in the mucous and submucous coats of the stomach, invading the pylorus as the process advances, causing a marked contraction and thickening of the entire organ with almost complete closure of the stomach outlet. In some of these cases the mucosa is so involved as to give many of the symptoms of atrophic gastritis with marked anemia. consecutive change in the secretory, motor and absorptive functions of the stomach. The organ is small, may present as a globular tumor, holds but little fluid. The use of the stomach tube gives valuable information. In some of these cases it is impossible, because of the thickened stomach wall, to inflate the organ. The differentiation of these cases presents great difficulty. They often resemble infiltrating cancer of the stomach.

(*b*) *Localized fibrous thickening of the pylorus,* likely to be annular, is occasionally found following previous conditions which may have given rise to but few symptoms and which were not dependent upon preceding ulcer. In these cases the ring-shaped constriction causes con-

secutive dilatation with its attending symptoms. It is not easy to palpate this small thickening of the pylorus in the majority of cases.

(3) *Hypertrophic stenosis, as described by* Cruveilhier and recently considered in detail by Boas, occasionally presents for diagnosis and treatment. We have seen this condition only at rare intervals, and where suspected we have not always felt that the diagnosis was correct. These cases are supposed to follow chronic gastritis with ultimate dilatation, in which the muscular and submucous tissue as well as the mucosa are finally sufficiently changed to give rise to constriction of the pylorus.

(4) *Stenosis dependent upon ulcer.*—Ulcer at or near the pylorus and in the first part of the duodenum with associated spasm, marked swelling which may be acute or due to secondary inflammation is unquestionably an occasional cause of pyloric stenosis. The pain is likely to be most severe as the food is ready to leave the stomach. An examination of the stomach contents materially aids in reaching satisfactory conclusions. In these cases the pain is out of all proportion to other complaints, there may be hemorrhage, with many other symptoms, subjective and objective of ulcer.

(5) *Acute inflammatory stenosis associated with infection, deep-seated gastritis and acute poisoning,* presents a train of symptoms coincident with these acute conditions, yielding as the associated inflammation subsides unless the changes are sufficiently deep to give rise to permanent lesions.

(6) *Syphilitic stenosis,* as described by D'Ursi and mentioned by others who have written on specific stomach diseases, is occasionally found with clinical features which do not permit of differentiation from other forms of cicatricial or inflammatory stenosis, save as we get authentic histories and, better still, results from antisyphilitic treatment.

(7) *Tuberculous stenosis* is occasionally associated with far-reaching disease of the stomach. Individual clinical experiences with *this* complication will always remain limited.

(8) *Stenosis dependent upon non-malignant growths,* fibromyoma, adenoma and polyp, described by Bouveret, Cornil and Chiari, only rarely found, may prove puzzling; the diagnosis can be made during life only after exploratory operation.

(9) *Stenosis due to distortion or traction,* giving rise to acute pyloric obstruction (Kussmaul, Albrecht and Riegel) dragging at a point in the horizontal part of the duodenum by the weight of the stomach contents where the former merges into the vertical duodenum, firmly fixed against the spine, is relieved at once by emptying the stomach with the tube or by free vomiting. This condition is occasionally responsible in all probability for acute and alarming dilatation.

Extrapyloric Stenosis.—It is difficult to divine all of the unusual and unexpected conditions which may lead to extrapyloric organic stenosis. We never cease to wonder at these pathologic possibilities. Clinical and pathological experiences in this field are rarely repeated. Perigastritis, gall-stone disease, adhesions to neighboring organs, particularly to the liver and gall-bladder, distortions due to innumerable anomalies and pathologic changes invading the organs already mentioned, the pancreas and kidney, may produce narrowing of the pylorus, consecutive dilatations and all of the symptoms of pyloric stenosis. In a paper written a number of years ago on the "Vagaries of Gall-stones," the writer reported a case in which monstrous gall-stone masses with associated adhesions caused all of the symptoms of pyloric construction during an entire year. At the end of this time the hard tumor disappeared, for a few days symptoms of complete intestinal obstruction followed, preparation was made for operation, when nature came to the rescue. Gall-stones resembling cobbles in size were passed by the rectum and the patient, a woman advanced in life, made a perfect recovery and is still living, without a return of symptoms.

In the majority of cases, extrapyloric stenoses are recognized, though the pathological condition giving rise to these may not be determined.

Perigastritis, peritonitis, perihepatitis, pressure due to pancreas tumors, large kidney masses and matting together of organs may be suspected as causes of extrapyloric stenosis.

In our study of the cases under consideration we must (1) give data which prove the presence of pyloric stricture, and (2) its benign nature must be established.

1. It is rational to suspect stenosis of the pylorus, whenever it is possible to demonstrate marked dilatation of the stomach with perceptible lowering of the greater and lesser curvature, recurring peristalsis from left to right (peristaltic unrest), which may or may not be painful, and may be and frequently is associated with ballooning of the organ; with either a palpable mass or thickening at the pylorus, more or less splashing and evidences still further of motor insufficiency, the latter made positive by means of the stomach tube, and such other refinements of diagnosis as are now used by all who study these cases closely which prove beyond peradventure the stagnation of food. *The fasting stomach found distended and containing food remnants must always arouse suspicion.* Stomachs may be abnormally large yet true ectasia with motor insufficiency may be absent. Megalogastria does not mean motor insufficiency or the inability of the stomach to perform its functions with fair satisfaction. Mechanical obstruction implies motor insufficiency sooner or later and this means inability of the stomach to empty itself with all attending subjective and objective symptoms. Stenosis present, the dilatation which is secondary remains, it is a permanent deformity. A stomach which is able to empty itself, but for some reason becomes dilated (true primary dilatation), does not remain permanently dilated as a rule. It is irrational to conclude that all enlarged stomachs are insufficient and that their pylori are stenosed. This physical condition must be associated with a knowledge of disturbed function before accurate conclusions are justified. Pyloric stenosis creates a decided disproportion between the stomach's task and its ability to perform. "Wo

Arbeitslast und Arbeitskraft (as Riegel so prettily expresses it), einander nicht mehr, wie in der Norm, das Gleichgewicht haben." This fact explains all of the conditions consecutive upon mechanical obstruction at the stomach outlet. Alkaline contents are as a rule hurried through the stomach and, contrary to general belief, far-reaching change as in achylia gastrica shows no tendency to gastrectasia. We must always be suspicious, where there is motor disturbance with hypersecretion and hyperacidity, of existing stenosis. In these cases it may often be difficult to decide, in spite of evidences of stenosis and dilatation, whether the former is the cause of the hypersecretion. Not all benign stenoses are associated with hypersecretion.

One of the leading symptoms of stenosis is vomiting. Frequency and quantity of vomited matter vary in accordance with the size of the stomach and the degree of constriction. In advanced cases the quantity of vomited matter is as a rule greater than the quantity of food and liquid taken. The vomited matter with obstruction, shows the presence of food long since taken. Vomiting relieves these patients, a load has been removed, it is wonderful to see how encouraged they are, while this enormous pocket is filling only to empty itself again. The reaction of the fluid vomited, its appearance, odor and color depend entirely upon the nature of the underlying disease. Associated with this vomiting at intervals, which may indeed be considered the leading symptom of stenosis, is marked change in appetite, sometimes perverted, fickle or completely lost. Intolerable thirst with symptoms of stenosis, in spite of the fact that the stomach is receiving fresh water for its relief, is always suggestive that this is of high degree. In these cases there is monstrous gastrectasia.

As the secondary changes follow, the tongue becomes glazed, often dry, coated at times, or remains unchanged. Constipation is the rule. The general appearance of the patient suffers, malnutrition, malassimilation are soon shown with all the other ear marks of the disease

Peristaltic unrest, recognized if the abdomen

is sufficiently watched, even during the early stage of the disease, becomes a symptom of great value. Peristaltic unrest may continue with marked stenosis until the patient is far advanced in the disease, when the musculature of the stomach wall becomes so feeble as to refuse longer to make the effort to empty itself, or when, with degeneration and infiltrating disease, it finally ceases. Often, in cases of stenosis, peristalsis is materially increased or aggravated during the early stages by the administration of soda and tartaric acid as carbonic acid is released. Splashing is usually present; to be of value it must be associated with other symptoms and we must make sure that it is due to retained stomach contents. As a rule these patients are able to produce the characteristic sounds without the assistance of the physician. Physical examination of the stomach including usual methods, often gives valuable data and must be practiced to confirm subjective symptoms.

The stomach tube as a diagnostic agent is indispensable. It is necessary in order to gauge the degree of obstruction and the amount of insufficiency to know the length of time food is held in the stomach. In none of these cases are we to neglect the inflation of the stomach, either through the tube or by carbonic acid liberated by the administration of a sufficient quantity of sodium bicarbonate and tartaric acid. Simple as is this maneuver we may occasionally misinterpret the result. I recall a case in which inflation filled the abdomen with what was considered a dilated stomach, in which the colon was distended through a gastrocolic fistula. The case proved to be one of infiltrating malignant disease of the stomach communicating with the colon as a result of ulceration and adhesive localized peritonitis.

The urine presents changes which cannot be ignored. Its quantity is reduced, it is concentrated, likely to be alkaline and contain indican.

Tetany is a symptom of very great value in the diagnosis of gastrectasia dependent upon any of the causes enumerated. In spite of all that *has been* written on this subject the characteristic

cramping and other alarming conditions in the majority of cases, this symptom is not always correctly interpreted and the average physician, because of its comparative infrequency, fails to appreciate its significance. I make this positive statement because of the very painful experiences which I have had in consultation practice. Symptoms referable to the nervous system, particularly neurasthenia, are frequent and predominate in cases of benign stenosis.

The heart, as the disease advances, may become ungeared, may partake of the general neurotic condition of the patient or may simply show increasing weakness with advancing disease. The presence of tumor positively connected with the pylorus or duodenum and symptoms of gastrectasia gives positive information.

If there is no tumor it may be difficult to decide between primary and secondary dilatation. In both conditions the subjective symptoms may be the same. In both we have seen peristaltic unrest and even bile in abundance mixed with the stomach contents with marked organic obstruction. In many of these cases, where repeated examinations of the stomach contents and close observation of physical signs have failed, we have applied the therapeutic test for purposes of diagnosis. We need not hesitate to subscribe to the statement of Mintz, Bouveret and Bevic that this often furnishes the only positive diagnostic means. Regulated diet, lavage of the stomach, alkalies in proper doses to meet underlying hyperacidity will, in primary dilatation be followed by material improvement, long periods of relief and partial return of motor sufficiency, as is shown by the tube and other tests. The alkalies probably relieve associated spasm. Such improvement with even temporary disappearance of symptoms we have never noted with true organic stricture of the pylorus, whether benign or malignant. It must be remembered that the quantity of retained ingesta and secretions are less in primary than in secondary dilatation, also that primary dilatation may recur with renewal of original symptoms. The presence of bile in large quantities with evidences of

constriction would indicate its location below the papilla in the duodenum. The neurasthenic element is much greater in cases of primary than of secondary motor insufficiency.

2. What are the distinctive clinical features which justify the conclusion that the stenosis is benign?

Previous History.—Whatever the suspicions, there is no stronger link in the diagnostic chain than the knowledge gained by painstaking inquiry into the previous histories of these patients. In both men and women the incidents of childhood and puberty as well as the suffering of mature life demand most thorough investigation. It would be an insult to your intelligence if I were to rehearse the data which such study reveals. Included are the clinical pictures of the many primary conditions which have finally led to stenosis. Patients find no difficulty in recalling these; they cover, as a rule, long periods of suffering which have left indelible impressions. A patient with final stenosis and gastrectasia depending upon ulcer has never allowed the sequence of symptoms to fade from his memory; neither has the patient who suffers from perigastritis, far-reaching adhesions with gall-stone disease, forgotten the early or late attacks. Time and patience only are required to hew a way out and through this quarry to the open light. Our results are sufficient to reward the effort. Failures in diagnosis, particularly of the conditions under consideration, are too often the result of hurry and lack of attention to detail.

The *age* of the patient is an important factor. The more advanced in life, the more likely is the stenosis to be malignant. This rule has its exceptions but they are few. Malignant disease presents a much shorter clinical history. While this may occasionally have latent periods, it is the exception. Chronicity with a long train of symptoms argues against malignancy. The symptoms of malignant stenosis are as a rule continuous, there is less likelihood of long periods of relief or marked periods of exacerbation. The wasting of the patient, the increasing

muscular enfeeblement go hand in hand. The blood conditions are entirely different. If there is associated anemia and this is out of proportion to the cachexia, with evidences of dilatation, we are not as a rule dealing with carcinoma.

Heredity must always play an important rôle in the etiology of pyloric cancer. Benign constriction with palpable pylorus presents a much smaller tumor than does cancer. Whether this thickening is due to intra or extra pyloric involvement, there is never such characteristic nodular feel nor is there such suggestive hardness and resistance nor is the mass usually as large as in cancerous disease. Physical examination of neighboring organs and distant glands gives data of great importance. Distant glands as well as those in the abdomen need thorough investigation. Thus Troisier, several years ago, and Pitt in a recent article in the *Practitioner* (April, 1905), have called attention to the enlargment of the glands above the clavicle, particularly behind the left sternomastoid, in pyloric and stomach cancer. Rapidity of growth is characteristic of malignancy. The use of the stomach tube, the examination of the stomach contents, chemical and microscopical, give information which the evenly balanced clinician will add to such other facts as have been made clear by thorough study. It will frequently be possible to recognize by these added physical methods early organic change at the pylorus. Much has been said of the value of free HCl. in the diagnosis of benign and malignant disease of the stomach. I feel that I have no right to repeat on this occasion the results of chemical analysis of the stomach in detail which furnish us with facts for differentiation. There are, however, a few facts which it may be wise to give to the profession in connection with a paper such as is here presented. They are the following:

Stomach contents which show gradual loss of HCl. from time to time, with the addition of organic acids as the free HCl. disappears, evidences of fermentation, must lead to a strong suspicion of malignant disease. It occasionally happens that cases of pyloric cancer continue

to show free HCl. to the end. Gradually fading hydrochloric acid, according to Jaworski may be due to the transition of non-malignant to malignant disease as well as to the gradual development of achylia gastrica in cases of mucous gastritis with more or less stenosis at the pylorus. This same change may also take place while malignant disease develops in the scar of an old ulcer and in the included tissue of a non-malignant ulcus ventriculi. Gluzinski makes the following statement: "If it were possible to recognize the transition of an acid gastritis into the mucous form and if, at the same time, it were possible to recognize that this change is due to the formation of a neoplasm and not to the simple cicatricial healing of an ulcer, then it would be possible to make an early differential diagnosis of malignancy." To accomplish this, Gluzinski makes three examinations of the stomach contents, one following the other on the same day. He first examines the remnants of the stomach contents early in the morning. The stomach is thoroughly emptied, chemical and microscopical examinations are made. The organ is then thoroughly washed, egg albumin is given; this is expressed after three-fourths of an hour. At noon beefsteak is given to be followed by the final emptying four hours later. These examinations are repeated several times at intervals of two or three days. If remnants of food are found in the stomach after fasting the question arises whether this is due to the simple atony of the stomach wall or mechanical obstruction, and we are thrown back to the same necessities of observation as before. Gluzinski places great importance upon the fact that with benign stenosis, particularly associated with ulcer, and its cicatrization, we find free HCl. present in varying quantities in all three examinations. It may be assumed that if at the first test, food remnants are found, at the second there is absence of HCl. or only faint presence, and after the beefsteak test marked reduction or complete absence of HCl. with lactic acid and bacteria, the stenosis may be considered malignant. If we find blood with the stomach contents in the wash water

with particles of tumor, the diagnosis becomes easy. Lactic acid may be present with achylia gastrica, associated with chronic gastritis, especially when these conditions have more or less stenotic accompaniment. In pernicious anemia, lactic acid is sometimes present with gastrectasia. Here the blood picture must differentiate. It is a rule which cannot be too often repeated that with malignant disease the cachexia is out of all proportion to the anemia, whereas, in those forms of pernicious anemia with gastrectasia and atrophy, the anemia is out of proportion to all other symptoms.

The, valuable reports which have reached us from Cohnheim's clinic, many of which are repeated in a paper recently published by Ackermann and Gompertz, which deals with the microscopical examination of the fasting stomach contents and its diagnostic value, when put into practice, added to the knowledge gained by chemical examination, give strong evidence which justifies more accurate differentiation during the incipient stages of the conditions under consideration. The microscopical examination of the stagnant stomach contents with the presence of HCl. shows, in cases of hyperacidity and hypersecretion that the muscle fiber is partially digested, that the striae have more or less disappeared, that starchy food is not changed. Yeast cells are found in the stomach contents with stagnation. Observers find these in chains and when the stenosis is benign these are accompanied by sarcinæ. Sarcinæ are not found unless the stomach contents contains HCl. Hence, their presence argues, as a rule, in favor of benign stenosis save in exceedingly rare cases of ulcus carcinomatosis. In this latter condition, the test of Gluzinski added, will give valuable information and as the disease advances, if malignant, there will be a gradual decrease of HCl. and sarcinæ; finally, both will be absent when the HCl. is found replaced by lactic, acetic and butvric acids, the sarcinæ by the Oppler-Boas bacilli. The microscopical appearance when the disease is malignant shows quite a different picture. Cohnheim asserts that "about 99 per

cent. of the cases with stagnation and Oppler-Boas bacilli or lactic acid are pyloric carcinoma, the remaining one per cent. being either gastritis gravis or a carcimona in the vicinity of the pylorus." Ackermann and Gompertz in their résumé make the statement that "Constant presence of pus, blood and possibly infusoria in the fasting stomach contents is absolute evidence of extrapyloric carcinoma." Still further, "Benign obstruction can be diagnosed early by the finding of sarcinæ, yeast cells in chains, or food remnants." "The early diagnosis of malignant obstruction of the pylorus can be made by the finding of the Oppler-Boas bacillus." There is no pathognomonic symptom of cancerous stenosis.

Large quantities of indican in the urine, an abnormally high urea output are always suggestive of malignancy. The more we deal with the diagnosis of these conditions, the more convinced are we that no other organ requires longer observation than does the stomach, before reaching conclusions which justify diagnosis. I know of no better dictum to place before the student and the hesitating practitioner than that of Leube to which we may unhesitatingly pin our faith in the differentiation of the conditions under consideration. I do not apologize for repeating it here. "Cancer of the stomach is present in all probability if the unquestionable stomach disease has attacked a patient between fifty and sixty years of age who formerly digested all food well. If such a person becomes dyspectic without an evident etiological factor, it may be assumed with considerable certainty that he is developing a gastric cancer." If this fact were better appreciated there would be a large addition to the credit side of accurate and early diagnosis.

Treatment.—When an internist, as conservative as was the late lamented Riegel, makes the following statement, "Gastrectasia following stenosis must be relegated to the field of surgery; in these cases operative interference alone leads to cure," any consideration of medical treatment *would seem* almost unnecessary. Our surgical

friends have established the fact that surgery scores its greatest successes in the treatment of the conditions under consideration, and it is these cases that the internist must refer to them for permanent relief with encouraging and increasing confidence. Our labor will end in cases of extreme benign stenosis as soon as the diagnosis is made. There will always be cases, however, in which for some good reason, either because of moderate stenosis with the hope of ultimate compensatory hypertrophy and a sufficient gastric function, because of inherent weakness in congenital cases, complications in the adult contraindicating operation, or the refusal of the patient to submit to radical treatment, the physician will be expected to relieve symptoms and make the lives of these patients as comfortable as his art can make them.

Prophylaxis.—Little need be said on the subject of prophylaxis, for little can be done. It is wise to warn patients with ulcer and chronic gastritis against carelessness and it will be the duty of the physician to give such directions as will lead to lasting cure, including the avoidance of food bolting, overeating, dissipation, giving at the same time thorough instruction in such hygienic measures and methods of living and eating as lead to an improved resistance. Further, we must warn the patient against the overloading of the stomach after long periods of restricted diet or after conditions which have led to inanition. The following indications for the medical treatment of benign stenosis of the stomach seem justified:

1. *Such foods only to be taken as the dilated stomach with motor weakness can prepare to be forwarded beyond the point of resistance for further digestion.* The volume of food must be small, concentrated and thin. Diet in these cases can only be rationally prescribed when we depend for our indications upon the degree of stenosis and the absolute or relative weakness of the muscle. The indications are offered by the free use of the stomach tube, the examination of the stomach contents early and late, and the microscope. Foods must be given with a thorough

understanding of associated secretory anomalies. It is far better to allow these patients to eat at short intervals, giving but little at a time, than to overload the stomach with the ordinary three daily meals. The quantity and quality of food can be easily determined if the physician will give this matter sufficient time. Experience teaches that in cases of extreme dilatation thirst is by no means relieved by the inordinate use of liquids. Von Mering has again emphasized the fact that fluids are not readily absorbed by the stomach. In these cases the stomach is still further distended by the excessive use of fluids which serve simply to deplete the patient still further by determining an extra supply of blood to the organ, thereby increasing hypersecretion, aggravating nervous symptoms and occasionally provoking tetany. On the other hand, solid or dry food refuses to pass the obstruction. It is, therefore, wise to pursue a safe middle course, giving foods in concentrated mushy liquid form. For this purpose such liquids should be chosen which will serve to carry other articles of food readily digested and absorbed. Thus bouillon, chocolate, cocoa and milk in small quantities may serve as vehicles for eggs, meat solutions, stale bread, plasmon, somatose, Mosqueras' beef meal, tapioca, arrowroot, rice and many other articles of diet which will occur to the physician as he studies the case. The various gruels and beef juice properly prepared, meat essence, Leube-Rosenthal food, scraped beef, broiled fish, sweet bread, custard, omelet, moderate amount of butter, milk toast, Zwieback with milk, potato puree, spinach and squash are all borne in most cases. There are but few cases associated with hypersecretion and hyperacidity in which food passes the pylorus, aided by such methods as we have at our command, which do not bear scraped raw beef well and to this, with eggs and liquids in small quantities the writer has often pinned his faith with encouraging results. A moderate amount of coffee at breakfast is allowable in the majority of cases. Stimulants are contra-indicated save in extreme conditions. These patients need to be kept under perfect control. For

this reason sanatorium or hospital treatment offers most encouragement. In many functional stenoses the use of olive oil or the emulsion of sweet almond oil, as recommended by Cohnheim, serves not only as a food, improving the general condition of the patient, but it seems to relieve spasm and friction. Rectal feeding is sometimes necessary.

2. *Seek to prevent stagnation of food with associated fermentation and the formation of organic acids.* This can best be done by the use of the stomach tube. In these cases lavage does yeoman's service. In the majority of advanced cases the stomach should be washed out twice daily, its contents siphoned at various depths, the tube introduced deep enough to relieve the stomach. Sufficient water should be used. A thorough knowledge of the shape and position of the stomach offers valuable indications for the successful use of the tube. Clean warm water without the addition of drugs is sufficient. Both in functional and organic cases the final flushing with cold water at a temperature of 50° F. seems to exert a tonic influence over the stomach and its musculature, in cases where the stenosis is not extreme. In order to get good and lasting results, this part of our work must be thoroughly done. Carelessness and undue haste or the empiric use of the tube can lead only to indifferent results. *Rational diet founded upon the knowledge gained by the use of the stomach tube and the microscope, with thorough lavage, become the most telling factors in the medical treatment of benign pyloric stenosis.* The accidents following daily lavage in these cases have been so few as not to discourage the treatment which most of us have found fairly successful.

3. *Assist so far as it is possible in the emptying of the distended stomach.* The methods already suggested with cautious massage, room gymnastics, the use of electricity (to which too much confidence has been given by some), with thorough attention to details and the use of drugs, such as shall be mentioned, are all that can be done to meet this indication.

4. *Prevent tetany and other nervous and distant symptoms.* Overcome neuropathic tendencies and conditions in functional stenosis leading to spasm. The writer is fully convinced that he has seen cases of tetany in which symptoms have been postponed by the timely emptying of the stomach. At the present time he has under observation a patient who refuses surgical interference, with benign stenosis, monstrous dilatation, hypersecretion and hyperacidity. Tetany can be promptly produced by the neglect of thorough lavage. It matters not what theory we adopt concerning the production of tetany, the fact remains that the majority of cases are associated with monstrous dilatation, residual stagnant food and hypersecretion. All facts warrant the prevention of such accumulation. Methods which overcome neuropathic tendencies and conditions which lead to functional stenosis require a thorough consideration of all that these cases present, attention to hygiene, exercise, diet and much detail which must of necessity occur to the thinking therapeutist.

5. *Improve the general condition of the patient and hold this at par or above par whenever possible.* This includes all of the methods already considered with such tonic treatment, moral suasion, change of air and scene as are indicated by the individual cases.

6. *Treat associated symptoms as they arise by rational and natural methods, including the use of drugs, attempting by these to overcome or counteract the effect of secretory and motor anomalies.* These indications cover a broad field of activity. Guard against pernicious activity. There is a strong tendency in the treatment of these conditions toward polypharmacy. The use of the alkalies and such remedies as act upon the secretions of the intestines and their musculature causing free movement of the bowels which tend to remain constipated, are indicated in many cases. Repeated tests give indications for the administration of alkalies or acids. With hypersecretion and hyperacidity we have recently had excellent results from the use of suprarenal extract and belladonna, combined with mechanical

treatment and alkalies. With peristaltic unrest and functional stenosis, belladonna, gelsemium and cherry-laurel water have frequently relieved symptoms. Eserine and the remedies above-mentioned have relieved associated flatulence and belching of gas. Strychnine, nux vomica and the bitter tonics have in many cases served to tone the patient and relieve symptoms. Spasm has sometimes been relieved by the use of small doses of atropine, the faradic current as recommended by Kussmaul; only rarely have we resorted to codeia. Recently, encouraged by an article from the pen of Haas-Brunn in the March number (1905) of *Die Therapie der Gegenwart* on "Eumydrin, ein Atropin-Ersatz, in der Therapie der Magen- und Darmkrankheiten," we have used the drug with some success. In no case has it been used long enough to offer positive conclusions. Resorcin, bismuth salts, benzonaphthol, Carlsbad salts and physol, made according to Merck's formula, may be used for the relief of troublesome symptoms.

With thiosinamine recently suggested, to be used hypodermatically (10 per cent. glycerin and water solution) and with the use of scopolamin the writer has had no experience. The galaxy of remedies suggested in our text-books proves conclusively that there is no single remedy upon which we can rely for the treatment of symptoms associated with stenosis of the pylorus. Rational and natural therapy, made as simple as possible, will prove most satisfactory.

[Reprinted from THE MEDICAL NEWS, July 8, 1905.]

ETIOLOGY, DIAGNOSIS AND TREATMENT OF BENIGN STENOSIS OF THE PYLORUS.[1]

BY GEORGE EMERSON BREWER, M.D.,
OF NEW YORK.

IN response to an invitation from the President of this society, I shall present for your consideration and discussion this evening, a brief review of our present knowledge of the etiology, diagnosis and treatment of benign obstruction of the pylorus.

In accepting this invitation, I realize fully that I am addressing an audience made up largely of expert observers in this line of work. I also recognize the fact that my own experience in gastric surgery is a comparatively limited one. I may, therefore, be permitted to state at the outset that my reason for presenting this subject before this Society is not that I hope to enlighten its members by anything new or original, but simply to bring this important topic before you for your critical discussion, which cannot fail to be of value to myself as well as to the profession at large.

Although dilatation of the stomach due to pyloric stenosis has been known as a pathological entity for centuries and its association with vomiting, delayed and painful digestion recognized in certain instances, it was not until the latter part of the nineteenth century that its importance began to be appreciated, and the differentiation between the benign and malignant forms of the stenosis made.

In 1868, Victor Bros, in his Strassburg thesis, reviewed the subject of benign obstruction of the pylorus and gave a classical description of the symptomatology of a typical case of the disease due to gastric ulcer.

The following year Kussmaul began the use of the stomach tube in the treatment of this condition and led the way for his pupils, Von de Velden, Leube, Ewald, Boas and a host of others, who studied the effects of obstruction on the function of digestion, and furnished valuable

1 Read at the Annual Meeting of the American Gastro-Enterological Association, April 25, 1905.

data for diagnostic purposes in the estimation of the motility of the organ and the chemical analysis of the gastric contents.

The first effect of a moderate narrowing of the pylorus is obviously an increased peristaltic effort on the part of the muscular apparatus of the stomach, to propel the food into the duodenum. This in an otherwise healthy individual generally results in a compensatory hypertrophy of the gastric musculature, and while this compensation is maintained no untoward symptoms occur. Sooner or later, however, if the disease is progressive, this compensation fails and a certain quantity of the food is habitually retained in the stomach. As in the case of the urinary bladder, this inability of the viscus to empty itself gradually results in dilatation of the organ with atony of its muscular walls. Stagnation of the gastric contents results in fermentation—fermentation gives rise to the formation of acrid acids and gases, which in turn irritate the mucosa and eventually cause a subacute gastritis or catarrh of the stomach.

Relief to the overburdened and irritated stomach can only occur from vomiting. This takes place at first at infrequent intervals, later more regularly, and when the obstruction becomes practically complete, after each meal.

As the stenosis increases a progressively diminishing amount of food passes into the intestine, and as absorption of water from the stomach itself is practically *nil,* thirst, diminished secretion of urine, constipation and rapid emaciation occur. When the obstruction becomes complete, death from starvation is the inevitable result.

The foregoing description applies to a progressive stenosis resulting in complete obstruction. In certain instances, however, the disease advances up to a certain point and then becomes arrested. In these cases the symptoms will vary according to the degree of obstruction, and the muscular tone of the organ. In general it may be stated that a stomach which is unable completely to empty itself over night, will surely give rise to symptoms requiring treatment.

In considering the etiology of benign stenosis

of the pylorus, we should, I think, follow the suggestion of Mayo, who maintains that lesions in the first two and a half inches of the duodenum should be included, as the pathological conditions producing stenosis in the portion of the duodenum above the papilla are practically identical with those of the pyloric region, and the resulting symptoms so similar that differential diagnosis is in most instances impossible except by exploratory incision.

The commonest cause of benign stenosis of the pylorus is undoubtedly ulcer. Gastric ulcer occurs at or near the pylorus with great frequency, most authorities agreeing that at least 75 per cent. of the lesions are found in this region. That duodenal ulcer is also of frequent occurrence is evident by the statistics of Mayo, who reports that the first portion of the duodenum was involved in 27 per cent. of his operated cases of ulcer.

A gastric or duodenal ulcer may produce a primary pyloric stenosis, either by the presence of an acute inflammatory exudate in the surrounding tissues or at a later stage by marginal induration or cicatricial contraction. Not infrequently when the process involves the deeper tissues or where a minute perforation has occurred, a circumscribed or spreading peritonitis is developed, which, if recovery takes place, results in the formation of adhesions which give rise to stenosis from angulation or fixation of the pylorus to the other organs.

Obstruction of the pylorus by spasm, caused by the irritation of an ulcer in some other portion of the stomach is said to occur and to give rise to the same train of symptoms as organic stenosis, but as Stockton has pointed out, too much reliance must not be placed on this statement which at present rests only on an hypothetical basis.

Diseases of the gall-bladder are second in point of frequency in the causation of pyloric stenosis. The commonest lesion of the gall-bladder to produce this effect is cholecystitis with local peritonitis. This gives rise to secondary adhesions which produce angulation and fixation of the pylorus or duodenum in the same manner as the adhesions from a perforated ulcer.

Fistulous openings between the gall-bladder and stomach or duodenum may occur, as a result of suppurative cholecystitis. Through these openings large stones may occasionally pass into the intestine. The presence of these stones may, very rarely, give rise to obstruction, whereas the cicatricial contractions of the fistulous tract and the perigastric adhesions more frequently cause narrowing of the pylorus and duodenum.

A case illustrating the degree to which narrowing of the pylorus and duodenum may reach from such a condition is the following:

Case.—A man, fifty-four years of age, had numerous attacks of pain and tenderness in the right upper quadrant of the abdomen. Later he had gastric distress and vomiting. These symptoms increased rapidly in severity and he became emaciated, weak and anemic. When first seen by the writer he stated that he had lost fifty pounds in the previous two months. The abdomen was very much retracted and a tumor was felt in the pyloric region which did not move with respiration. Stomach enormously dilated, no free hydrochloric acid present; hemoglobin, 40 per cent.

On opening the abdomen, the gall-bladder, pylorus and duodenum were found fused together by dense adhesions. The gall-bladder was opened and found to contain a quantity of pus and seventeen stones. When these were removed, the examining finger passed through a narrow opening into a secondary cavity also containing numerous calculi. We at first thought we had to do with a double gall-bladder, but after a long and difficult dissection, it was found that the supposed accessory gall-bladder was in reality, a pouch formed by the first portion of the duodenum into which the suppurating gall-bladder had emptied. This cavity was separated above from the stomach and below from the second portion of the duodenum by two large, almost complete cicatricial strictures, each intimately associated with the inflammatory induration around the margin of the fistulous opening. On laying open this duodenal pouch *thirty-six* stones and a moderate amount of foul-*smelling pus* were evacuated. A pyloroplasty by

the Finney method was performed, but the patient succumbed to the shock of the prolonged operation. In this case the occlusion had been practically complete for a number of weeks.

The presence of benign tumors at or near the pylorus, has occasionally been reported as giving rise to obstruction. The writer on one occasion found and removed a small adenoma from the anterior wall of the duodenum just above the papilla. Lipomata arising from the submucous layer of the stomach, near the pylorus, have occasionally been reported, some reaching a large size. Myomata papillomata and lymphadenomata have also been found but are exceedingly rare and as a rule too small to produce serious obstruction. A few instances are on record of a polypoid tumor with long pedicle, which would occasionally be carried into the pyloric orifice and produce intermittent occlusion.

More important than the preceding group are the hypertrophic stenoses. This condition has been described by a number of observers including Tilger, Boas, Habershon and Oettinger, and each has given it a different name. Practically all unite, however, in regarding the obstruction as one of hypertrophy and sclerosis of the muscular and submucous coats, causing great narrowing and rigidity of the orifice. The pyloric region is often so thickened that a distinct tumor may be felt through the abdominal wall.

Three varieties of the affection have been described: First, the congenital stenosis, in which there is thickening of the submucous coat with hypertrophy of the circular and longitudinal muscular fibers, but without lesion of the mucous membrane, obviously a non-inflammatory condition; second, the adult type which appears to follow a chronic gastritis in which the mucous and submucous coats of the entire stomach are involved, giving rise to a cirrhosis of the organ, and third, where the disease seems limited to the pyloric region. In the cirrhotic variety the capacity of the stomach is greatly reduced and dilatation is impossible, while in the third variety, dilatation is generally present.

The importance of syphilis as an etiological factor in the production of pyloric stenosis has

been variously estimated by different authorities. Engel reports a syphilitic history in 10 per cent. of all his ulcer patients. Stockton has reported several in which the symptoms have yielded promptly to antisyphilitic treatment, and numerous autopsies have demonstrated beyond a doubt that gummata and typical syphilitic ulcers may exist at or near the pylorus and give rise to more or less serious obstruction.

Among the rare forms of pyloric obstruction may be mentioned foreign bodies, masses of hair, wool or other non-absorbable substances which have been swallowed and rolled into large balls by the muscular movements of the organ, also benign tumors of the liver, gall-bladder ducts or pancreas pressing on the stomach or duodenum, enlarged lymph nodes and possibly kinking of the duodenum from gastroptosis or movable kidney. Mayo has described a valvular formation due to inflammatory shortening of the gastrohepatic omentum.

In considering the clinical history of pyloric obstruction it must be borne in mind that we may and frequently do have two distinct groups of symptoms; first, those due to the disease which gives rise to the stenosis, and second, those due to pyloric narrowing, the resulting gastrectasia and the chemical changes which occur in the retained gastric contents. The first effect of pyloric narrowing is retardation in the normal emptying of the stomach after an ordinary meal. This may give rise to no subjective symptoms, and the only indication of such retardation may be that furnished by the use of the stomach tube which shows that the viscus still contains a portion of the ingested food at a period when it should be empty. Riegel states that after an ordinary meal, the stomach should be found empty in seven hours. In mild degrees of motor insufficiency, food will still be found in the stomach seven hours after a meal, but not in the early morning after a light supper the previous evening. In general the degree of motor insufficiency may be estimated by the amount of food residue found at various times after the taking of a test meal.

Occasionally patients with mild degrees of *motor insufficiency* complain of a sense of weight

and discomfort in the epigastrium after a heavy meal but not when a moderate amount of food is taken. There is, as a rule, no actual pain and the symptoms disappear as soon as the stomach is empty. In more advanced cases of stenosis, where compensation is present, the peristaltic movements of the stomach can occasionally be seen or felt through the abdominal wall. This may be associated with a subjective symptom which Kussmaul has described as *peristaltic unrest*. When the stomach begins to dilate and fermentation takes place, the patients complain of a greater degree of fulness and epigastric pressure after each meal. Eructations occur, which may be odorless or foul, from the presence of sulphureted hydrogen. Not infrequently heartburn is present from the occurence of fatty acids in the fermenting gastric contents.

Of all the symptoms of pyloric obstruction, however, vomiting is the most important and characteristic. It may occasionally occur in the earlier stages of motor insufficiency before dilatation has taken place, but when gastrectasia and fermentation are developed it is more or less constantly present, and in the most advanced cases it occurs after each meal.

It is characterized by the large amount of food expelled at one time and the fact that the vomited matter often contains particles of food taken the day, or several days, before. As a rule the vomitus is acid in taste and smell, and often foul from decomposition. When allowed to stand it generally separates into three distinct layers—the upper of froth, the middle of a grayish fluid and the lower of solid particles of food.

As vomiting gives relief in the majority of instances, patients not infrequently get into the habit of inducing it whenever the gastric distress is severe. In advanced grades of pyloric obstruction, the patients suffer greatly from lack of fluids. This gives rise to intense thirst, dryness of the skin and mucous membranes and a marked diminution in the excretion of the urine. Crámer is accustomed to estimate the degree of dilatation by the quantity of urine excreted, dividing his patients into three classes, the mild when 1,500 c.c. or more is excreted in twenty-four hours,

those of medium severity when the urinary excretion is reduced to 1,000 c.c. and the severe cases when the excretion is 500 c.c. or less.

Loss of weight necessarily begins as soon as the stenosis reaches such a degree that the requisite amount of food is not forced into the intestine, it is usually progressive and in the end emaciation is extreme. Progressive anemia is present in all of these cases, the degree generally corresponding with the rate of emaciation.

In the later stages of gastrectasia patients generally complain of loss of appetite, headache, mental torpor and various paresthesias. In a certain number of cases tetany will develop, evidenced by the occurrence of tetanic contractions in the hands, arms and legs, with occasional trismus and transitory blindness. This frequently follows severe vomiting or an attempt to pass a stomach tube, and unless gastric drainage is at once instituted, the disease proves fatal in a large proportion of the cases.

With the exception of those cases where a tumor can be palpated in the pyloric region, there are no physical signs which help us to a diagnosis in the early stages of benign stenosis. When gastrectasia occurs, however, physical examination frequently furnishes important data. Inspection will often reveal the outlines of a dilated stomach through a thin abdominal wall. Occasionally the greater and lesser curvatures can be palpated, and splashing may often be elicited at a time when the stomach should normally be empty. Distending the stomach with air or water will often enable us by percussion to identify its limits and thereby form a fairly reliable estimate of its size. In general it may be stated that with the lesser curvature in its normal position, when the greater curvature can be demonstrated at or below the umbilicus, dilatation is presnt.

The foregoing description applies only to instances of uncomplicated benign pyloric stenosis. It is perhaps the exception to find adult cases which present such a typical clinical history, as the symptom complex in the majority of instances is modified by the occurrence of some preceding or accompanying pathological condition which gave rise to the stenosis.

In those cases due to ulcer therefore we are apt to have a history of digestive disturbance long before the signs of pyloric obstruction occurred. In the majority of instances such patients complain of more or less acute attacks of epigastric pain after eating, often with vomiting of acid material at the height of the attack.

They have generally restricted their diet because certain articles of food seemed to produce more distress than others, and as a result are frequently emaciated and anemic. There may be a history of hematemesis or melana.

In those cases in which the stenosis is due to the late contraction of the scar of an ulcer, or to peritoneal adhesions due to ulcer, there may be a distinct interval between the symptoms of ulcer and those of pyloric obstruction. In the great majority of cases, however, the history would indicate that the early symptoms of obstruction were due to the induration surrounding an open ulcer, and in these instances the cardialgia, eructations, epigastric tenderness and vomiting overshadow for a time the comparatively mild symptoms of early motor insufficiency. Under these conditions the pain is often more severe than in ulcer without obstruction, due to the mechanical irritation of the ulcer by the increased muscular action of the stomach to force the food into the intestine. While the pain is frequently limited to a small area in the epigastrium which is often exquisitely tender, it may radiate to the back, the breasts, to the neck or hypochrondriac regions. The suffering in these cases is often extreme until relieved by vomiting of an abundant acid material.

Hyperchlorhydria is present in practically all of these cases, as demonstrated by gastric analysis. Not infrequently there is in addition a more or less continuous secretion of gastric juice evidenced by morning vomiting of a cloudy acid fluid containing a few flakes of solid matter. Later when dilatation has occurred this morning vomiting contains remnants of food taken the evening before.

As in other cases of gastric ulcer, the symptoms may undergo marked variations in intensity,

owing to care exercised in the diet and the employment of rest and other remedial measures.

It may happen that the symptoms of ulcer may cease entirely, leaving only those of a progressive gastrectasia; on the other hand the pain may persist throughout the disease to the end. In the later stages of the affection, the appetite which in the beginning with an abundant hyperchlorhydria was exaggerated, almost invariably is lost and there is instead a disgust for food of any kind.

In those cases of obstruction due to adhesions from gall-bladder suppuration, there is usually a history of attacks of pain in the upper abdomen, which, however, do not exhibit the same relation to the taking of food as in gastric ulcer. In these attacks the pain and tenderness are located chiefly in the right hypochondriac region and the vomiting when it occurs is mostly at the beginning of the attack. There is no evidence of hyperchlorhydria. In addition there is generally a history of one or more severe attacks accompanied by fever and other evidences of local peritonitis. The occurrence of jaundice is occasionally mentioned.

As the symptoms of gall-bladder and duct disease are practically always intermittent, with long intervals of quiescence, the presence of pyloric obstruction and gastrectasia, is generally indicated by the occurrence of more or less typical symptoms of this condition which, however, may at any time be interrupted by those of an acute gall-stone colic or attack of cholecystitis.

Although it would be quite impossible in the limited time at my disposal to review the symptomatology of all of the rarer lesions giving rise to pyloric stenosis, I desire to call attention briefly to one of the varieties of this malady which has not received from the profession at large the attention it deserves.

Congenital stenosis of the pylorus was first described by Beardsley, of New Haven, in 1788. Meltzer, in the *Medical Record* of July 20, 1898, reviews the literature of the subject and gives the clinical history of a case under his care, together with the appearances found at operation

and the pathological report of the specimen obtained at autopsy.

Dorning has recently reported another case with typical symptoms, in which he was able by palpation to detect a pyloric tumor during life. He stated that at the time of his report, about sixty cases had been recorded. Meltzer states that the life history of these cases may be divided into three phrases: First, the period of simple muscular insufficiency; second, an attempt at compensation, and third, the stage of atony and progressive dilatation.

The clinical histories of the reported cases show a striking similarity, and correspond to the three periods described by Meltzer.

At first the child nurses with avidity but soon regurgitates the meal. After several such attempts the mother generally finds that if a small feeding only is allowed it may be retained. Vomiting may therefore be infrequent for a few weeks after birth, the frequency depending largely on the degree of stenosis. During this period the child is generally restless between feedings and shows evidence of constant thirst. Sooner or later the stage of atony and dilatation occurs and the vomiting becomes more frequent and exhausting. The child grows progressively weaker, emaciates and may develop stupor and other nervous symptoms. At this period the stomach can generally be demonstrated to be dilated and flaccid, the urine is scanty and constipation is marked.

The use of the stomach tube is of great value in confirming the diagnosis in these cases. The presence of food in the stomach two hours after a feeding, when associated with the above symptoms, is sufficient to establish the diagnosis.

In considering the treatment of benign pyloric stenosis it seems hardly necessary to say more than a few words to an audience the members of which appreciate so keenly the fact that the cause of all the untoward symptoms of this disease is purely a mechanical one, and can be relieved only by reestablishing a normal connection between the stomach and intestine. It would be a waste of time even to consider drugs, massage, electricity and lavage, which at best can only be regarded as palliative measures. With the

exception of the few instances in which the obstruction is so light that the symptoms can be easily relieved by dietetic regulation, or in those exceedingly rare cases where the stenosis is due to a syphilitic ulcer or gummatous deposit, and which quickly respond to anti-syphilitic treatment, surgery and surgery alone can offer relief to these patients. In perhaps no other class of lesions of the operations upon the alimentary canal have the results of surgical intervention been more strikingly satisfactory.

It will sometimes be possible to remove the causes of the stenosis by operation, such as peritoneal bands or adhesions, new growths or enlarged lymph nodes pressing upon the pylorus or first part of the duodenum, foreign bodies or benign tumors of the stomach wall. In the majority of instances, however, it will be necessary either to enlarge the contracted pyloric orifice by plastic operation or to create a new opening between the stomach and small intestine. Digital dilatation of the pylorus has been abandoned by surgeons.

The original pyloroplasty of Heinecke-Mikulicz is gradually giving way to other more radical procedures for the reason that recurrences after its employment were frequent. The operation of Finney and posterior gastro-enterostomy are the two procedures employed at present for the relief of pyloric stenosis. Finney's operation which consists in enlarging the pyloric opening by a plastic operation which unites the first portion of the duodenum with the adjacent wall of the stomach is the operation of choice in cicatricial stenosis where the tissues are not too much infiltrated, in all other cases gastro-enterostomy is to be preferred. The technic of these procedures is so well known that a description of it here would be superfluous.

As the mortality of these operations in capable hands, where the physical condition of the patient is still fairly good, should be below 4 per cent. in benign cases, and as a permanent cure may be expected in the majority of cases, it is difficult to appreciate why the rank and file of the profession should not give their unqualified approval to this method of treatment and why it is not more generally practised.

DISCUSSION.

DR. S. W. LAMBERT, of New York City: In discussing the diagnosis of benign stenosis of the pylorus, as compared with malignant stenosis, too much stress cannot be laid upon the fact that the history of the patient, rather than the physical signs, will make up the principal factors in the case. No matter what the character of the stenosis may be; no matter what the element that caused the obstruction of the pylorus or of the upper part of the duodenum may be, I think the results of a test-meal examination, the symptoms of the stenosis, the accumulation of food, etc., are practically the same whether the obstruction be benign or malignant. The only exception, perhaps, is in those cases where a true peptic ulcer is the cause of a cicatricial contraction. In such cases we should get the history and the symptoms of the preceding ulcer, including the characteristic hyperchlorhydria.

In going over the histories of these cases of benign stenosis at the New York Hospital, I have found three which illustrate the the point I wish to make. One of these was that of a woman, 27 years old, who had complained of gastric pain for three years and vomiting for one year. She had lost 70 pounds in weight. A test-meal showed the presence of lactic acid and an absence of free hydrochloric acid. She was operated on and the pylorus was found to be hard and sclerotic. There was no tumor. The operation of pyloroplasty was done in November, 1903, and up to January, 1905, she had gained fifty pounds in weight, and considered herself in perfect health.

The second case was a woman of 50, who for the previous six months had suffered from gastric distress and vomiting, with considerable loss of weight. There was a palpable tumor in the epigastric region, about half the size of an adult fist. An examination of the stomach contents showed an absence of free hydrochloric acid and the presence of lactic acid. A gastroenterostomy was done in January 1902, and the diagnosis made of an irremovable carcinoma. In spite of this the patient was enjoying excellent health in November, 1903, and had gained 40 pounds in weight. Her stomach tumor was still palpable, and the examination of the gastric contents was the same as before the operation. In January of the present year her condition was still the same, and she considered herself in perfect health. The tumor in this case was doubtless a myoma.

The third case came into the hospital with a general peritonitis, and a diagnosis was made of a perforated duodenal ulcer. An operation was done in October, 1903, and the patient recovered. The following January the patient returned with all the symptoms of a stenosed and dilated stomach. An examination of the stomach contents showed an absence of free hydrochloric acid, and the presence of lactic acid. A gastroenterostomy was done to relieve the post-operative stenosis, and the patient left the hospital, cured, in March, 1904

In all these three cases, one, a benign sclerotic stenosis of the pylorus; the second, a myoma, and the third a post-operative pyloric stenosis, the the analysis of the stomach contents pointed to carcinoma. The previous history of these patients, however, threw more light on the conditions which existed than did the physical examinations, including the examination of the gastrc contents.

So far as the question of treatment goes, I believe in surgery as the only possible help. I have found that pyloroplasty gives equally good results as gastroenterostomy.

DR. HOWARD LILIENTHAL, of New York City: This subject has been so thoroughly discussed that there is nothing to be said excepting perhaps a word or two about the surgical technique.

It seems to me that the less shunting and switching we do, and the fewer artificial plumbing arrangements we make for the relief of benign stenosis of the pylorus, the better. I believe that the simplest form of gastroenterostomy, which Dr. Brewer has illustrated, and which I am in the habit of performing, is the best one, and I do not believe that the others are necessary.

Gastroenterostomy had probably better not be perfomed in cases of benign stenosis if one can possibly manage to restore the parts involved to a condition more closely approaching the normal, and pyloroplasty, preferably by the Finney metheod, is my operation of choice in these cases. It is most applicable when nothing is left of active disease, and only the cicatricial stenosis remains. The operation is a very beautiful one. It gives us a pylorus which cannot re-contract, and relieves the symptoms more perfectly than any gastroenterostomy can do, because the pylorus is in its normal place, where the most active peristaltic motion occurs.

I have operated upon seven cases of benign stenosis by the Finney method. Five of these were referred to me by Dr. Morris Manges, one by Dr. Nathan E. Brill, and one by Dr. J. Rudisch. Five of these cases made a perfect recovery; two died. One of these deaths was due to an accidental infection, resulting in peritonitis; the other patient bled to death into his own stomach, without a single attack of vomiting. The opening from the stomach into the duodenum, is so large that vomiting after the operation is almost impossible. When the stomach contracts, its contents take the path of leats resistance, which is into the bowel.

I believe that pylorectomy will in the future be a much more common operation in benign stenosis than it has been in the past. It is not necessary to do a wide pyrolectomy, as in cancer, but when you are in doubt as to the character of the lesion, it is well to attempt not only to relieve the symptoms, but also to remove all the doubtful tissue. As our technique becomes more perfect the operation will become less dangerous, and the statistics will probably compare favorably with those of the Finney operation. Of course, it should only be done where there is a palpable tumor on operation.

DR. EDWARD QUINTARD, New York City: The subject has been so thoroughly threshed out that I can add very little to the discussion.

I am of the opinion that we should be very careful before we resort to operative procedures in any of these cases, because I have seen many cases of partial pyloric stenosis recover under medical treatment. I would be the last to advocate waiting when it is necessary to call in the surgeon; invoke the aid of surgery too early rather than too late should be our rule, yet we repeatedly see cases of benign pyloric stenosis, especially partial stenosis, recover under medical treatment. It is my practice wherever a stenotic condition is suspected, to put such patients to bed and then to make a thorough investigation of the case before calling in the surgeon. Rest in bed accomplishes wonders in many of these conditions and greatly aids in clearing up the diagnosis.

Dr. Brewer mentioned stenosis of the duodenum, and stated that it might give rise to the same symptoms as stenosis of the pylorus. I have seen stenosis of the jejunum cause absolutely the same symptoms as stenosis of the pylorus.

I thoroughly agree with Dr. Lambert in reference to the presence of lactic acid in these cases. I have come to place less reliance upon the presence or absence of lactic acid in the stomach secretions, as a means of diagnosticating between benign and malignant stenosis. We cannot depend upon any one symptom, but must take the entire history of the case. The more carefully we study the patient's history, the fewer mistakes we shall make in our diagnosis.

The possible occurrence of a peptic ulcer after gastroenterostomy is another reason why I hesitate to pass my cases over to the surgeon. I cannot help but fear the consequences of operative interference in many of these cases.

I agree with Dr. Lilienthal's statements regarding pyloroplasty, and from what I have seen of the operation I should certainly recommend that method in preference to gastroenterostomy, if the latter operation can be avoided in any way.

DR. A. G. GERSTER, of New York City: In my remarks on this subject, I will confine myself largely to a brief account of my personal experience. In medicine, as in other lines, conservatism is commendable only when its tendency is not the conservation of a malady. Caution ought to be tempered by courage. But speaking in general, I make it an inflexible rule not to operate upon a case of benign stenosis of the pylorus until it had been thoroughly demonstrated that other methods of treatment had been and are ineffectual.

In considering the advisability of operationg on a patient, much depends on the personal equation. A condition which to some is bearable might be to others absolutely intolerable. A hard and fast line cannot be drawn, but our main guide should be the amount of suffering experienced by the patient.

I have requested Dr. A. A. Berg, adjunct of my division at Mt. Sinai Hospital, to collect the material comprising benign stenosis of the pylorus operated upon since 1899. It consists of the histories of 23 cases, upon which the following procedures were done:

	Total Cases	Cured	Died
*Pylorectomy, for calous ulcer and benign stenosis	1	1	
Posterior gastro-enterostomy, for open ulcer and benign stenosis.	15	12	3
Finney's gastro-duodenostomy, for benign stenosis	2	2	
Heinecke-Mikulicz pyloroplasty, for benign stenosis	1	1	
Heinecke-Mikulicz operation on contracted gastro-enterostomy opening	1	1	
Division of peripyloritic adhesions causing benign stenosis	2	2	
Posterior gastro-enterostomy and secondary entero-enterostomy..	1	1	
	23	20	3

CAUSES OF DEATH—Two patients were moribund at time of operation from repeated gastric hemorrhages, for the control of which all internal measures had failed. One patient was in extremis at time of operation from acute dilatation of the stomach, secondary to pyloric stenosis from chronic perforated ulcer.

*This patient was, four months after discharge, readmitted with diffuse perforative peritonitis, due to peptic ulcer of the jejunum, and died.

DR. J. KAUFMANN: I will confine my remarks to the indications for the surgical treatment of benign pyloric stenosis. I think I am on record as being very much in favor of surgical interference in certain gastric conditions, but I must say that I cannot indorse the radical view of Dr. Elsner and Lambert, that the surgical treatment is the only one for benign pyloric stenosis. The surgical treatment is not without certain disadvantages, as shown by the specimen which I demonstrated today. In that instance a gastro-enterostomy was done with excellent results, but it eventually led to complications which caused the patient's death. Mikulicz says that his experience with peptic ulcer has made him very cautious in advising this operaton, and he now limits it to those cases where it is absolutely necessary.

Dr. Gerster says, that we should learn to distinguish between those cases that really need surgical interference, and those that do not. I think we must not look for indications for surgical interference to the degree of stenosis only, because that symptom is sometimes very misleading. We may have to deal with a tumor caused by an inflammatory process, or to a spasmodic condition, which may readily disappear under appropriate treatment. Kussmaul has published such cases. His publication on

this subject was really the starting point for the whole modern pathology of the stomach, and was based on the experience gained by the medical treatment of benign pyloric stenosis. His cases were further advanced than any we see at the present day, and some of these cases recovered under medical treatment. I have frequently seen patients recover from their symptoms of pyloric stenosis under appropriate medical treatment. A more important indication for surgical treatment than the degree of the stenosis—if not too pronounced and permanent—is the suffering caused by the primary disease which led to the stenosis. We see f.i. patients with gastric ulcer suffer for years from very severe pains, who have only a slight degree of stenosis, mostly caused by pylorospasm. In those cases surgical treatment often removes the obstacle, which prevents the healing of the ulcer and thereby ends the suffering.

One word about the differential diagnosis between benign and malignant pyloric stenosis. I think the most reliable and pronounced symptom of malignant disease is that of marked bacterial fermentation.

DR. ELSNER: In connection with inflation of the stomach as a diagnostic factor, I call your attention to a case of infiltrating cancer of that organ in which there was a gastrocolic fistula, the result of ulcertation and protective peritonitis, in which it was supposed that we were dealing with an enormously dilated stomach, because on inflation that organ seemed distended, when in truth we were dilating the transverse colon through the fistula with the tube in the stomach. Such conditions may not often be present but the possibility of dilating the large intestine through the stomach must not be overlooked. In this case there was no fecal vomiting, no data which made the true pathologic condition recognizable. At the post-mortem the stomach was found infiltrated in all directions, no larger than an ordinary base-ball. The gastrocolic fistula was a surprise to all those who had seen the case. In connection with the cases reported by Dr. Lambert, it must be remembered that we often meet carcinoma of the stomach in which the disease remains latent during long periods. In fact, such behavoir of carcinoma ventriculi is not uncommon. We have seen a number of these cases during the past and have called attention to them at various times. Dr. Quintard's case of obstruction of the jejunum reminds me of a patient whom I saw during the past month, in which the diagnosis of probable pyloric obstruction had been made. A thorough investigation of the case proved it to be one of stricture of the jejunum and ileo-coecal valve. There was the history of occasional fecal vomiting and balooning of the intestines in the lower half of the abdomen. This of course excluded obstruction of the pylorus and made the diagnosis, because of the distension of the gut in unequal segments, very easy. If the paper which I presented is carefully read you will find that I favor surgical interference in all cases of extreme benign pyloric stenosis.

ONOMATOLOGIA GASTROLOGICA.*

BY A. ROSE, M.D.,

NEW YORK.

True men of science are anxious to see purity and correctness of medical language prevail among the writers and speakers of the day. In view of this fact the following suggestions may be acceptable in regard to some terms employed in gastrology. Fortunately there exists only a small number of incorrect names or solecisms in this branch of the medical sciences, but even some of these have caused confusion and should be eliminated. The countless and multiform conceptions contained in medical science require innumerable technical terms. Medical lexicons became a necessity when, for the purpose of the disseminating of scientific information, the useful custom had been established in medical literature, not only of giving the names of the diseases and anatomical names in the author's native language, but also of employing terms for diseases and anatomical conceptions which either were those of the old Greek and Roman physicians, or were compounded and introduced from the classical languages to denote new conceptions unknown to the ancient Greeks and Romans.

*Read before the American Gastroenterological Association, at the Academy of Medicine, New York, April 24 and 25, 1905.

In our surgeon-general's library is an *Onomasticon, sive Lexicon Medicinæ*, by O. Brunfels, printed in Strasburg, in the year 1534, a folio; and this is perhaps the oldest medical lexicon that appears in print.

With the introduction of more and more new compounds, more and more irregularities came into our onomatology, because no exact rules could be formulated to control the formation of new compounds which had to be constructed to some important extent from a dead language, i. e., as long as Greek was considered a dead language, an error which prevailed until very recently. In the case of Latin there exists no authority, that is no people speaking it as their native tongue, who can decide whether a new Latin compound is correct; and in the case of Greek, the Greeks, who really could decide, have not been, and as yet are not, considered.

In Greece itself countless demands are constantly made on the language, which is developing rapidly so as to keep pace with the many new political, scientific, technical, commercial and journalistic requirements of the day. The Greeks do not adopt, with the constantly imported foreign ideas, the connotative words also of foreign peoples. They refuse to have a hybrid language. Their history, their na-

tional pride, lead them to exclude foreign words and to take such necessary elements from the ancient Greek as enable them to create new symbols for new ideas. In order to demonstrate the impossibility of attaining absolute correctness in new formations derived from the Latin, we have to study the methods of the Greeks and the manner in which they go to work to preserve the purity of their language. When constructions and forms have been remodelled after the old Greek, incorrect elements, when discovered, are extirpated with more and more severity and tact. Before a new formation is introduced into the regular language it has to stand a severe test and criticism. Nothing will be accepted and grafted into the regular language which deviates in any way from the genius of the Attic tongue.

Comparing these Greek precautions against corruption of language with the wanton barbarism of our nomenclators, we are forced to the conclusion that the only way to purify our onomatology is to consult our Greek colleagues of the University of Athens and to follow their advice.

In the year 1821 was published a medical lexicon, by L. A. Kraus, in which the most frequently used names of Greek origin were enumerated, with the special object of exposing

incorrectness wherever it was noticed by him. This author speaks against the formation of hybrids, considering it a principal rule to be observed, to compound words only from one and the same language, that is, Greek with Greek and Latin with Latin. We learn by his lexicon that in the days of our grandfathers there existed word monstrosities which, thanks to the critics like Kraus, have since been eliminated. Some of these words would horrify you, although we have new hybrids which are as bad as those of olden times. Some which were exposed a century ago are still in use. I will not dwell now on the ideas of Virchow in regard to incorrect terms, or on the work of Hyrtl, in which he exposes grammatical inaccuracy, calling our onomatology incorrect, ridiculous, absurd, and hence unscientific; or on the attempt of Henle, or above all on the work of the men of the German anatomical society, to create a correct onomatology. I have before pointed out the vain exertions of all these excellent men and great scholars, and I have furnished conclusive evidence that they failed and all their labor was for nothing, simply because they ignored the existence of living Greek. All this I have exhaustively treated in several publications.

There are not many—by no means so many as in some other branches of medicine—gram-

matically incorrect or misinterpreted terms in the literature on gastrology, but such as do exist have caused serious confusion.

The mouth of the stomach is called "cardia;" this may lead to misunderstanding in some instances.

Let us keep in view that *gaster* is the belly; that we have to recognize this fact and not use the word "gastric," when we wish to refer to the *stomach* expressly.

Kymograph is incorrect; the word is kymatograph.

For megalogastria, let us better say megagastria.

For gastroptosis, gastroptosia.

There is no confusion to be feared by employing these incorrect terms, but whoever takes pleasure in the purity and beauty of language will find them intolerable.

In former editions of his work Riegel has given the following differentiation: 1. Simple atony or insufficiency of the stomach. 2. Atonic or typical ectasis, or dilatation. In the latest edition, however, he has corrected his error, and states that atony and insufficiency are not synonyms, and that every atony is also dilatation. I mention this because I have noticed that the former errors of Riegel have been admitted into an American book on diseases

of the stomach. Ectasia, which we so often see, should be ectasis, and notwithstanding English distionaries and the English custom of centuries, dilation and dilatation are *not* one and the same.

It is strange that that monstrosity of a word, gastrosuccorrhœa, about which so much complaint has been made, is still employed. Our grandfathers called it chylorrhœa. If we wish to be very exact, let us call it chylorrhœa gastrica, but by no means, as some have suggested, gastrorrhœa, for this would be another barbarism. We can add the word *rhœa* to a word meaning a liquid, like blennorrhœa, spermatorrhœa, galactorrhœa, etc., but the first word in the composition must not be the name of an organ. Words like metrorrhœa, otorrhæa, and gastrorrhœa are decidedly barbaric.

In an American book on disease of the stomach, from which I have quoted some of the foregoing errors, I find the following passage:

"It is impossible to invent a term which shall comprise and connote the important features of all types of motor and mechanical insufficiency; as clear a classification as any is one based on Riegel and Boas, as follows: Simple gastric atony, or motor insufficiency, or myasthenia without dilatation." As mentioned already, Riegel has changed his views;

he says distinctly, as I maintained years ago, that motor insufficiency is *not* atony; and I wish to repeat also that the word myasthenia is without any definite meaning. Our American author speaks of atonic dilatation, calls it even dilation. I may be permitted to quote another passage from him: "Gastric atony is a condition of reduced or lost tonicity of the musculature. It is a state of sub or hypo-tonicity, also very aptly designated as gastric myasthenia." Such complication, instead of saying plainly gastric atony, means abdominal relaxation. The barbarism, gastrectasia, is found very often. The preposition "ec" is impossible in the middle of a word. Let us say ectasis gastrica or gastric ectasis.

But not only are medical terms often barbaric because the nomenclators do not take into consideration living Greek, but all our dictionaries, in their attempts at the etymology of Greek words, will continue to spread errors so long as they do not consult living Greek.

Recently I found the Greek word asepsia quoted from a dictionary as being asepsis. This error is analogous to the famous "post partem" error. The Greeks say antisepsis, but asepsia, according to grammatical rules.

The mistakes that happen in translations from one language into another by the aid of

a lexicon are often a source of hilarity. We have all laughed over the witty pamphlet, English as She is Spoke, because we know better and can see the absurd incongruity of the errors; but to those who really know anything of the spirit of the Greek language—and that spirit is to-day exactly what it was in the days of the great classical writers—much of the modern would-be Greek medical nomenclature is just as absurdly incongruous. And surely, though we may not insist that every medical practitioner, as such, ought to be imbued with the Greek spirit, we have a right to demand that those who fashion our Græco-medical terms for us shall be imbued with that spirit. We are indebted to Dr. Einhorn for an example of "Medical Greek as She is Spoke," since he has named stagnation of the stomach caused by motor insufficiency, i. e., by atony or stenosis of the pylorus, "ischochymia."

Ἅγιος ὁ θεός, Ἅγιος Ἰσχυρός, Ἅγιος Ἀθάνατος ἐλέησον ἡμᾶς.

May the Almighty deliver us from such onomatology!

Ischuria, which he mentions as an analogue, is quite a different kind of retention. *Ischo,* which Dr. Einhorn cites as meaning "retain," means to check or arrest, actively, forcibly, by the exercise of a restraining power; not to retain from the lack of power to expel.

Whoever wishes to hunt up barbarisms in our general medical onomatology, however, will be better rewarded for his search in any other branch than in gastrology.

On April 18, 1787, Lavoisier presented at a public session of the Royal Academy of Sciences a memoir on the necessity of a reform of chemical nomenclature, which reform he wished to be considered as a national work. He reasoned as follows:

The onomatology furnishes the real instruments for the operation of the mind; it is important that these instruments should be of the best kind, and it is indeed working in the interest of science, for the progress of science, when we exert ourselves to improve our onomatology. Referring to the manner by which we acquire our knowledge in general, he points out the importance of a perfect onomatology for those who are beginning to devote themselves to the study of science. The logic of science essentially adheres to scientific language. Science cannot teach anything that is confessedly unscientific and false.

In science we have to distinguish three things: The series of facts which constitute the science; the ideas which recall the facts; and the words to express the ideas. The word

has to develop the idea, the idea has to embrace the fact; these are three impressions with the same seal. Since the words preserve the ideas and transmit them, perfection in science is impossible without perfection in language.

However true the facts may be, however correct the ideas developed by facts, only wrong impressions will be transmitted so long as the expressions by which they are communicated are not exact.

History tells of the great influence Lavoisier's nomenclature has had on the development of scientific chemistry; history also tells of the progress in botany due to Linnæus's nomenclature.

The words of Lavoisier referring to nomenclature in general and to chemical nomenclature as he found it—a regular jargon, the creation of the alchymists—apply in a great measure to many of the new formations in medical language in use at the present day, which are by on means construed according to principles pronounced by Lavoisier. It is time that we should be delivered from all barbarism in our onomatology..

Bergman, the great scientist, the pupil of Linnæus, who died in the year 1784, wrote during the last days of his life to M. de Mor-

veau, one of Lavoisier's cooperators, "do not spare one single improper term."

Reformation of medical onomatology can be accomplished, as I have pointed out on several occasions, without difficulty and without creating confusion. Some day I shall appeal to the medical profession at large to consider my suggestions.

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